

DISEASES OF THE ESOPHAGUS

ILLUSTRATED

J TERRACOL

Professor of the Faculty of Medicine of Montpellier, France

RICHARD H SWEET

Associate Clinical Professor of Surgery, Harvard Medical School

W B Saunders Company

PHILADELPHIA AND LONDON

1958

© 1958 BY W B SAUNDERS COMPANY *Copyright under the International Copyright Union* All Rights Reserved This book is protected by copyright No part of it may be duplicated or reproduced in any manner without written permission from the publisher *Made in the United States of America* Press of W B Saunders Company · LIBRARY OF CONGRESS CATALOG CARD NUMBER 58-7950

This work is dedicated to

THE DEVOTED CLINICIANS OF PAST AND PRESENT

who have contributed significant advances
in the management of esophageal disease

PREFACE

DISEASES OF THE ESOPHAGUS have assumed a position of great prominence in recent years. Conditions which were once overlooked or rarely identified only to be regarded as curiosities, about which nothing could be done, have become important in the experience of every physician no matter what his field of special interest and have found their rightful place both in diagnosis and in therapy.

This change of emphasis was brought about first by the invention of the techniques for accurate observation and diagnosis by means of esophagoscopy and roentgenology and secondly by the momentous advances of the past fifteen years in the techniques of anesthesia and surgery together with the discovery of effective agents with which to combat infection.

With these considerations in mind and the knowledge that there was no comprehensive authoritative book on the esophagus and its diseases in the English language I conceived the idea several years ago of undertaking such a work. In 1952, however, I discovered that the task had already been accomplished in the French language by Professor Terracol and his collaborators. Recognizing the excellence of this volume I abandoned my own project which was already in the formative stages. The inaccessibility to many English readers of the vast store of valuable information contained in Professor Terracol's book, however, seemed to me to be so unfortunate that the idea of translating it into English gradually took shape in my mind. As time went on it seemed that an American edition, revised and modernized to include some of the newer knowledge particularly regarding surgical treatment, would be of inestimable value to whoever might be concerned in any way with the diseases of the esophagus and their treatment.

Finally after consultation with the W. B. Saunders Co., who gave their enthusiastic support, Professor Terracol was approached about the proposal. It was with great pleasure and gratitude that his gracious consent and that of Masson & Cie the French publishers were received.

The subject of diseases of the esophagus has reached the stage where the combined efforts of both an endoscopist and a surgeon are necessary to bring

to bear all the available knowledge based upon experience which the subject requires. Thus the opportunity to collaborate with one who is as expert in the field of endoscopy as Dr. Terracol is particularly gratifying and important.

It was with great satisfaction and enthusiasm that this work was begun. Now that the task has been completed, I am more than ever convinced of the enormous value and importance of Professor Terracol's work. The American edition is offered in the hope that the reader of English will be able to profit as much from its perusal as its merit deserves.

For the benefit of those who may already be familiar with Professor Terracol's book, "*Les Maladies de L'Esophage*," a few comments about how the present volume was evolved may be interesting. This present work is based upon the second edition of the original book published in 1951. The project involved translation, rearrangement, revision, and the rewriting of some sections. Wherever possible the original text was followed rigidly, though the translation has been rendered into the English idiom. It has been my earnest endeavor to preserve both the flavor and the meaning of the original, but whether Professor Terracol has been as fortunate in this regard as his long ago predecessor at Montpellier, Francois Rabelais, was with his Thomas Urquhart remains for others to decide. The rearrangement of some of the sections of the original book was done in an effort sometimes to consolidate scattered material in one place and at other times because of personal preference regarding the sequence with which certain subjects are to be considered. Most of the material in the excellent separate section at the back of the French edition by P. Betoulières and P. Levrier on "The Radiologic Diagnosis of Diseases of the Esophagus" has been incorporated throughout the text under the appropriate headings.

Wherever possible in the interest of brevity, repetitious or controversial matter and in some instances historical discussions have been deleted, though with regret. Certain sections such as that on mega-esophagus have been shortened, others such as that dealing with the surgical treatment of carcinoma have been greatly expanded.

An entirely new section on esophageal substitution is included and the discussions of surgical treatment have been rewritten throughout the entire book, although most of the excellent drawings of operative technique, especially those in the sections written by Dr. Campo, have been retained.

After much deliberation the final section of the original work dealing with gastroscopy, though exceptionally thorough and interesting in itself, was deleted as being only related to the subject at hand because of the need for using the esophagus to reach the stomach. There is adequate mention, in Chapter 23, of the esophagosopic complications of gastroscopy.

The majority of the original illustrations have been retained although some have been deleted or replaced by new ones which are thought to be improvements over the old. Approximately 85 of the 408 illustrations of the present volume are new. I am indebted to my son Roger A. Sweet for the drawing which appears as Figure 13.

The bibliography of the 1950 Edition of "*Les Maladies de l'Esophage*" is so exhaustive that it is undoubtedly unique. In fact, no such comprehensive listing of references to the anatomy, physiology, roentgen and endo-

scopic investigation, and diseases of the esophagus can be found elsewhere in convenient form. For this reason, in spite of the fact that its inclusion adds considerably to the size of the book, it was decided not to delete any of this material which should be of inestimable value to the serious student.

In order to increase the cohesiveness of the book as a whole, however, the sections which are to be found in the French edition at the end of each chapter and subject, or in some instances scattered throughout the text, have been grouped together at the back of the book in the American edition. The arrangement of references according to chapters with appropriate division into sub-headings for the various subjects has been preserved in proper sequence.

No effort has been made to re-translate into English the numerous references from the English and American literature which appear in French in the 1950 Edition. The appearance of these titles in the French language should not present any serious obstacle to their utilization by anyone who may be likely to employ them.

It should be mentioned that an occasional reference may appear in that part of the text as translated from the French Edition which cannot be found in the bibliography. This is because Dr. Terracol, as explained in his preface to the Second Edition of his book, found it necessary to omit many of the titles from the older literature which were found in his First Edition. A perusal of the bibliography of the First Edition should make up this lack should anyone be interested, though much of this older material is now of relatively little value.

In order to bring the subject matter up to date, additional references are included at the end of each group of material in the French listing. The literature on the subject of the esophagus has become so voluminous that not all references could be included because of the limitations of space. A choice has been made on the basis of their relative importance and timeliness.

It would be impossible to convey adequately my feeling of gratitude to Professor Terracol for the privilege of writing this American edition of his valuable work and for his generous cooperation and liberality in permitting me to use my own judgment regarding the manner in which the task was to be done. His attitude has made it possible for me to make certain contributions of my own which make me feel that, although the work is principally his, it actually represents a collaborative effort which is exceedingly gratifying to me.

Because of the changes in the arrangement of material in the present edition it is difficult to mention the names of Professor Terracol's collaborators in appropriate places throughout the text as he has done. I have therefore taken the liberty of enumerating these contributions here as follows:

Betoulieres, P., and Levrier, P. Roentgen examination of normal esophagus and many of its diseases

Campo, A. Surgical treatment of congenital atresia and tracheo-esophageal fistula, double aortic arch and dysphagia lusoria, and carcinoma

Delmas, J. Anatomy (with Dr. Terracol)

Despons, J. Cicatricial stenosis (with Dr. Terracol)

- Guibert, H L* Microscopic anatomy
 Esophageal biopsy (with Dr Terracol)
 Benign tumors (with Dr Terracol)
- Guns, P* Physiology (with Dr Terracol)
- Harant, H* Fungus infections (with Dr Terracol)
- Mourner-Kulm, P* Abnormalities of veins (with Dr Terracol)
 Diphtheria (with Dr Terracol)
 Disorders associated with diseases of the skin and mucous membranes (with Dr Terracol)
 Agranulocytosis (with Dr Terracol)
 Communications with the air passages (with Dr Terracol)
- Sargnon, M* Malignant tumors (with Dr Terracol)
- Vialle, J, and Hashinger* Esophagoscopy (with Dr Terracol)
- Zeman, F G* Foreign bodies (with Dr Terracol)

Grateful acknowledgement is made to my secretary, Miss Dorothy N Saunders, for her valuable suggestions and assistance and for her persistence with the drudgery of typing the early drafts and the many subsequent revisions of various sections which were required before sending the finished manuscript to the publisher, and for her skill in reading the proof

My sincere thanks are due also to Mr John L. Dusseau and Mr Robert B Rowan of the W B Saunders Co for their encouragement and to Mr Dwight J Hotchkiss, also of Saunders Company, for his intelligent cooperation and valuable editorial supervision

I should like also to express my thanks to my associate, Dr E W Wilkins, Jr, for his kindness in reading the galley proof of the entire work and for his assistance in the revision of Chapter 2 (The Physiology of the Esophagus)

RICHARD H SWEET

Preface to the Second French Edition—1951

A FEW YEARS before 1940 Professor Belinoff of Sofia undertook a crusade in behalf of the esophagus throughout Europe and particularly in France. The esophagus said he that organ about which so little is known.

Belinoff is dead and it is regrettable that he should have died before the marvelous evolution of esophageal surgery. This effort is the work of the great technicians of the United States. One must not forget, however, in France the first attempts of Quenu, of J. L. Faure of Fougue and of Hartman and of many others abroad. None of these however no matter how great his technical virtuosity had at his disposal the present-day techniques of resuscitation, closed circuit anesthesia and above all, the now available antibiotics.

The systematic employment of all these methods not forgetting careful preoperative preparation and thorough attention to postoperative care assures immediate success in the vast majority of cases.

In spite of this, it seems at present that at least so far as cancer is concerned the end results which are all that count, have been rather disappointing. This must not be allowed to detract, however from the fact that surgery of the esophagus has become possible and that it has other indications notably in congenital malformations, cicatricial stenoses, ulcers and benign tumors.

In this second edition certain names in the first edition will not appear. Belinoff we repeat is dead. Physician-General Worms also a man of unusual intelligence and remarkable understanding, so also Peroni, the young hopeful of Italian laryngology and finally our friend Marcel Wisner. War always strikes in a cruel fashion.

We repeat our thanks to the original collaborators and to the new ones who have consented to join in our effort. So many new facts have accumulated in ten years that this second edition is more important than the first.

Several comments are in order. We have exerted every effort to assure the

PREFACE TO THE SECOND FRENCH EDITION-1951

unity of this work by a careful review of all the text, the elimination of repetition, and, what is more important, of contradictions

The bibliographic references concern only the past ten years. The reader is referred to the first edition for those concerning the earlier years.

The illustrations are numerous and representative. They have been accumulated from works of our colleagues to whom we are indebted, or from our own

J. TERRACOL

CONTENTS

Chapter 1	
ANATOMY GROSS AND MICROSCOPIC	1
Chapter 2	
PHYSIOLOGY	43
Chapter 3	
ROENTGEN EXAMINATION OF THE NORMAL ESOPHAGUS	52
Chapter 4	
ESOPHAGOSCOPY AND ESOPHAGOSCOPIC BIOPSY	80
Chapter 5	
INSTRUMENTAL EXPLORATION (BOUGIENAGE)	117
Chapter 6	
CONGENITAL MALFORMATIONS	123
Chapter 7	
ALTERATIONS IN POSITION CHANGES IN CONTOUR AND COMPRESSION	154
Chapter 8	
ESOPHAGOSPASM	173

Chapter 9	
MEGA-ESOPHAGUS (ACHALASIA)	199
Chapter 10	
ATONY AND PARALYSIS	256
Chapter 11	
DIVERTICULA	247
Chapter 12	
ABNORMALITIES OF THE VEINS	276
Chapter 13	
ACUTE AND CHRONIC NONSPECIFIC ESOPHAGITIS	292
Chapter 14	
ESOPHAGEAL ABSCESS, PHLEGMONOUS ESOPHAGITIS, PERIESOPHAGITIS, AND MEDIASTITIS	308
Chapter 15	
NONSPECIFIC ULLERATIONS	320
Chapter 16	
SPECIFIC INFECTIONS	333
Chapter 17	
DISORDERS ASSOCIATED WITH DISEASES OF THE SKIN AND MUCOUS MEMBRANES	352
Chapter 18	
ACUTE CORROSIVE ESOPHAGITIS (CHEMICAL BURNS)	358
Chapter 19	
CICATRICAL STENOSIS (STRICTURE)	376
Chapter 20	
ESOPHAGEAL SUBSTITUTION	397

Chapter 21

ACQUIRED COMMUNICATIONS WITH THE AIR PASSAGES (ESOPHAGOTRACHEAL AND ESOPHAGOBRONCHIAL FIS- TULAE)	419
---	-----

Chapter 22

RUPTURE AND EXTERNAL INJURY	435
-----------------------------	-----

Chapter 23

INSTRUMENTAL PERFORATIONS (ENDOSCOPIC INJURIES, ETC)	443
---	-----

Chapter 24

FOREIGN BODIES	456
----------------	-----

Chapter 25

EXTRACTION OF FOREIGN BODIES FROM THE ESOPHAGUS	469
---	-----

Chapter 26

BENIGN TUMORS	490
---------------	-----

Chapter 27

MALIGNANT TUMORS	502
------------------	-----

Chapter 28

SURGICAL TREATMENT OF CARCINOMA OF THE ESOPH- AGUS	535
---	-----

Chapter 29

IRRADIATION TREATMENT OF CARCINOMA OF THE ESOPH- AGUS	575
--	-----

Appendix Diets and Tube Feedings	591
----------------------------------	-----

Bibliography	597
--------------	-----

Index	671
-------	-----

CHAPTER I

Anatomy. Gross and Microscopic

PART I GROSS ANATOMY

¹THE ESOPHAGUS is a pliable easily dilated tubular organ comprising a prolongation of the funnel-shaped hypopharynx. It traverses from above downward the neck, the posterior mediastinum, the diaphragm and a short space within the abdomen to join the stomach at the cardia. Its superior limit or mouth lies at the level of the inferior margin of the cricoid cartilage along the lower border of the cricopharyngeal bundle of the inferior constrictor muscle of the pharynx, beneath which the recurrent laryngeal nerve disappears. In a man standing erect with his head in a normal position this level is opposite the body of the sixth cervical vertebra. With the head fully extended it rises to the level of the body of the fifth cervical vertebra and, contrariwise with the head lowered in complete flexion it descends to the level of the seventh cervical vertebra. In women the superior limit of the esophagus is slightly higher.

Because of the variations of these relationships it should be pointed out that the single exact relation of the upper orifice of the esophagus is to the tubercle of the cricoid cartilage. Actually an imaginary line drawn horizontally along the inferior border of this tubercle separates the pharynx from the esophagus, no matter what the position of the head may be.

[The inferior limit of the esophagus at the cardia is usually opposite the left side of the body of the tenth or eleventh thoracic vertebra.]

One should realize however, that these anatomical relationships cannot be precisely defined because of the great mobility of the esophagus in general and because of variations among individuals. It should be kept in mind therefore that the terms mouth of the esophagus and cardia employed to designate the upper and lower ends of the organ are applied to regions which are in fact transitional zones subject to individual variations, and are useful only as names

General Characteristics

¹ Because of its ability to expand in a circumferential direction, the distensibility of the esophagus is considerable. The tissues of the esophagus are normally fragile, its wall is thin, and disruption may occur easily. This is particularly so in pathological conditions, such as caustic burns, cicatricial stenoses, and neoplasms. In many of these conditions, instrumentation either with the esophagoscope or with bougies may easily produce a rent through the entire thickness of the wall above the obstruction. Furthermore, the esophagus tolerates poorly the presence of foreign bodies, not only those with sharp points which may perforate its wall easily, but also those which are blunt ²

Finally, from the surgical viewpoint the esophagus is a tenuous organ which supports sutures badly and in which a limited incision may, if not handled with care, be accidentally torn beyond the limits considered safe or desirable for the procedure to be carried out.

Along the course of the esophagus between the fixed points at the cricopharyngeal region and its termination in the stomach, there are numerous attachments. These are of two kinds: muscular attachments such as the inferior constrictor of the pharynx, the tracheo-esophageal muscle, the left broncho-esophageal muscle, and the phreno-esophageal muscle, and fibro-elastic attachments such as the peritoneal, vascular, and ligament-like attachments to the vertebral column. In spite of these attachments, the organ maintains its mobility which is, however, subject to the effects of respiration, coughing, and the pulsations of the heart and the aorta. The mobility of the organ is also influenced by the position of the head which brings about a lengthening and tensing of the esophagus when extended, and, conversely, a shortening and increased mobility in flexion. With the exception of the two fixed points at its pharyngeal attachment and at the diaphragmatic hiatus, the esophagus is capable of undergoing limited changes of position in the anteroposterior plane and somewhat more extensive changes in the transverse direction.

This relative ease of movement is necessary to insure the proper functioning of the organ during deglutition. As soon as its mobility is impaired, dysphagia appears. During every examination of the esophagus with the esophagoscope, areas of abnormal fixation should be sought for with care ³

Direction

⁴ The course of the esophagus does not follow a straight line. It is in close relation with the vertebral column throughout much of its length and as it passes through the neck and emerges into the thorax the organ lies far posterior. At the level of the fifth or sixth thoracic vertebra, where it passes beneath the left main bronchus, it begins to assume a forward inclination. This is gradually increased as the esophagus descends through the lower mediastinum, the esophageal hiatus of the diaphragm, and the upper abdomen to the point where it joins the stomach.

In the transverse plane the esophagus lies directly in the midline throughout its cervical portion and in the superior mediastinum. After its passage behind the left main bronchus and the aortic arch, however, it deviates sharply to the left. It is for this reason that, during esophagoscopy, in order to visualize the

cardia the esophagoscope should be directed along a line extending from the right angle of the mouth to the anterior superior spine of the left ilium. In fact, from the esophagoscopic viewpoint it is particularly important to realize that from above downward the esophagus tends to swing to the left. With the subject in the sitting position, however, the downward pull of the stomach tends to straighten this curve.

Tortuosities of the esophagus other than the gentle curves mentioned exist only in the elderly, in conditions in which the larynx assumes a lowered position, and in the cadaver as a result of the upward push of the diaphragm following postmortem gaseous distention of the abdomen.]

Length

In the average adult examined post mortem the length of the esophagus is approximately 24 to 28 cm, but extremes of 20 and 50 cm have been reported. Average lengths of the various segments are as follows:

Cervical portion	5 to 6 cm
Thoracic portion	16 to 18 cm
Diaphragmatic portion	1 to 1.5 cm
Abdominal portion	2 to 3 cm
	24 28 5

These measurements naturally vary depending upon age, sex, stature, and position of the body. Likewise, transitory variations depend upon such mechanical factors as the phase of respiration, coughing, and the gastric volume. All measurements are therefore approximate. Of interest to the esophagoscopist, though not so much to the surgeon, is the relative distance from the superior gingival margin (or the upper incisor teeth) to important landmarks. Figure 1

Age	Total length	Distance from upper jaw to		
		Mouth of Esophagus	Aortic Arch & Bronchus	Cardia
Children	cm	cm	cm	cm
9 days	10	7	12	17
3 months	11.5 12	7.5	12.5	19
14 "	12	10	14	22
21 "	13 14	10	15	23
3 years	14	10	15	23
4 "	15	10	15	23
5 "	16	10	17	26
6 "	17	10	18	28
11 "	18	11	19	28
15 "	18 20	14	23	33
Men		14-16 average 15	23 29 average 26	36 50 average 39.9
Women		12 15 " 13.9	22 27 " 23.9	32 41 " 37.5

FIGURE 1. Table of Von Hacker. Length of the esophagus and its segments calculated from the upper gingival ridge.

shows average measurements for children and adults of each sex for the length of the entire esophagus and for the distances to the mouth of the esophagus, to the region of the left main bronchus and aortic arch, and to the cardia. In practice, the study of roentgen films is helpful, but experience and the educated eye above all make the recognition of these landmarks possible.

Shape and Caliber

The esophagus is an ill-proportioned cylinder. Its shape varies from one region to another and, what is most important, some portions are narrowed in relation to other portions which are relatively dilated. Whether or not these areas of narrowing are the result of developmental abnormalities or of extrinsic pressure from adjacent organs is relatively unimportant. The fact is that in the living subject there are four principal constrictions: one located at the level of the cricoid cartilage, one opposite the aortic arch, one at the left main bronchus and one in the diaphragmatic portion.

These levels of constriction of the esophagus are of interest not only to the anatomist but also to the pathologist and in particular to the surgeon. Foreign bodies often lodge in these areas, and chemical burns, tumors and other pathological strictures show a predilection for these zones. At one time when the only means available for the exploration of the esophagus was the use of a sound or probe, surgeons made use of the momentary arrest of their exploring instrument at these regions as detected by touch to serve as orientation in the downward passage of the instrument. However, as the use of the esophagoscope became established for the investigation of the esophagus in the living subject, it was recognized that the presence of these areas of relative constriction was not as important as the purely anatomical studies of previous years had indicated. In fact, it has become more and more obvious in recent years that purely anatomical studies static as they are, can be of little value as compared with the ever-increasing importance of the study of function. With this in mind a consideration of the areas of constriction of the esophagus from the physiological standpoint makes it clear that they are not all of equal importance. The question then arises as to whether some, if not all, of these zones should not be regarded as regions of dynamic muscular activity in the wall of the esophagus instead of fixed regions of anatomical narrowing. This question has been raised with regard to the retrocricoid constriction at the mouth of the esophagus and particularly with the narrowing of the esophagus at the point where it passes through the diaphragm. Some authors believe an actual sphincter may exist at this point, although such is not the case. This question is given greater consideration on pages 19-20.

The *cricopharyngeal constriction* is a narrowing produced by the cricopharyngeal muscle and the cricoid cartilage. It is the narrowest portion of the esophagus and the point most feared by the esophagoscopist because of the difficulty of passing the esophagoscope through it. It lies approximately at the level of the sixth cervical vertebra. The average caliber at this level as given by Chevalier Jackson is 23 mm. in the transverse and 17 mm. in the anteroposterior diameter.

The *aortic constriction* is found at the point of contact of the esophagus with the arch of the aorta, where the compression of the vessel against the wall of the esophagus can normally be seen. This area of narrowing lies at the level of the fourth thoracic vertebra. Its transverse diameter averages 23 mm and its anteroposterior diameter 19 mm.

The *bronchial constriction* lies at the point where the left main bronchus crosses in front of the esophagus, which is somewhat compressed behind it. Certain authors (chiefly anatomists) have confused the bronchial and aortic constrictions and have spoken of them together as the broncho-aortic constriction. In the living subject, however, they must be considered separately because the bronchial constriction is more obvious than the aortic. The bronchial constriction lies opposite the fifth thoracic vertebra. In caliber it averages 23 mm in the transverse and 17 mm in the anteroposterior diameter.

The *diaphragmatic constriction* (so-called diaphragmatic pinchcock) is the narrow segment which occurs at the point where the esophagus traverses the diaphragm. It lies at the level of the tenth thoracic vertebra. In caliber it is approximately 23 mm in both transverse and anteroposterior diameters.

Three other points of constriction are sometimes seen. The first is the retrosternal, situated midway between the cricopharyngeal and the aortic constrictions and probably produced by the local compression of adjacent organs in this narrowest portion of the mediastinum. It is occasionally the site of lodgement of ingested foreign bodies. The second of these points of constriction is the cardiac, found where the esophagus passes directly behind the pericardium. This constriction is usually seen only when the right atrium is enlarged as in mitral stenosis, and is usually of no clinical importance. The third and rarest of these constrictions is a narrowing of the lower esophagus just above the diaphragm, produced in an elderly person by the anterior curvature of an unusually tortuous arteriosclerotic aorta. The presence of this condition is best recognized by roentgen examination. It may give rise to dysphagia in some instances.

Segments of the Esophagus

Pharyngo-esophageal Segment

The mouth of the esophagus, called by Chevalier Jackson the cricopharyngeal pinchcock, deserves consideration as an independent region which may be called the pharyngo-esophageal segment. Externally the posterior surface of this portion is covered by a perivisceral aponeurosis consisting of a large very resistant fibrocellular veil of tissue which is attached to the vertebral column and which comprises the anterior limit of a long expanse of loose connective tissue which extends from the base of the skull to the esophageal hiatus of the diaphragm. Beneath this membrane may be seen the pharyngo-esophageal muscle layer.

In gross appearance there is a striking difference between the musculature of the pharynx and that of the esophagus. The former is composed of obvious thick groupings of muscle fibers while the latter is thin and smooth. At their junction the esophagus seems to disappear beneath the overlying prominence

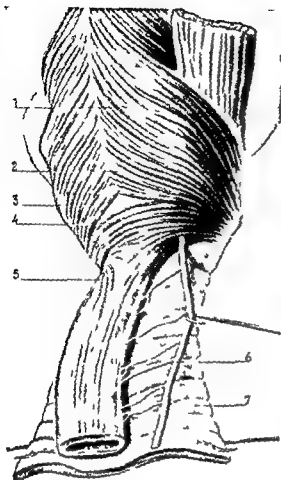


FIGURE 2 The pharyngo esophageal segment (exterior view with trachea opened and rolled out) Note the difference of direction of the muscle fibers of the pharynx as compared with those of the esophagus and the prompt fusion of the muscle fibers of the esophagus to accommodate themselves to its tubular shape also strength of the pharyngeal fibers as compared with the relative weakness of the esophageal fibers 1, 2 Oblique fibers of the inferior constrictor muscle, 3 upper weak point 4 cricopharyngeus muscle 5 lower weak point, 6 recurrent nerve, 7, trachea

of the lower pharyngeal musculature and the line of demarcation between the laryngopharynx and the cervical esophagus is sharp

Furthermore, the muscle fibers of the two organs assume different directions in this region. Those of the pharynx extend obliquely downward and laterally from the median raphe, while those of the upper esophagus extend upward and laterally. This arrangement of fibers outlines at the back of the junction of the pharynx and esophagus a lozenge-shaped formation, the center of which is demarcated approximately by the lowermost transverse fibers of the inferior constrictor muscle of the pharynx which inserts upon the cricoid cartilage to form a musculocartilaginous ring at the mouth of the esophagus. The location of this bundle of fibers can be recognized at esophagoscopy.

Between the ascending oblique bundles of muscle fibers there exists a weak point in the wall of the esophagus which is of great importance. This is the site of origin of the so-called pulsion diverticulum of the pharyngo-esophageal junction, which lies above the cricopharyngeus muscle (Fig 2). At this point, also the maladroitness of the esophagoscope may cause a perforation of the wall of the esophagus. If the false passage thus created is unrecognized, the instrument may actually be thrust into the superior mediastinum from this point. Similarly, beneath the lowermost pharyngeal muscle bundles (the so-called elevators of the esophagus) there is another hiatus or weak point (Fig 4).

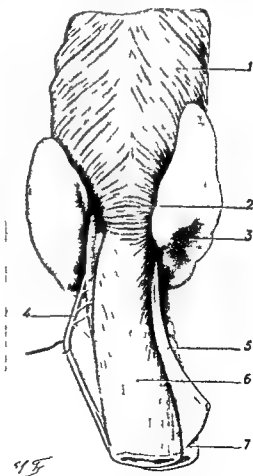
Thus the pharyngo-esophageal segment as viewed from the exterior com

prises a lozenge shaped region which is traversed crosswise by the cricopharyngeus muscle which demarcates clearly the separation between the pharynx proper and the cervical esophagus (Fig. 4)

Viewed from within, the pharyngo-esophageal segment is divided into two levels by a fold of mucous membrane called the hypopharyngeal ridge (Fig. 5). The upper level is the vestibule of the hypopharynx. The lower level comprises the retrocricoid narrowing of the esophagus. This hypopharyngeal fold is like a lip of the mouth of the esophagus. During esophagoscopy the end of the instrument usually impinges against this fold. Contraction of this fold is a manifestation of pharyngo-esophageal spasm. Flat foreign bodies often lie hidden beneath it. It should be emphasized that this fold or prominence is of a muscular nature, formed in fact by the cricopharyngeus muscle. It has nothing to do with the submucous venous plexus described by Elze and Beck.

In summary, the lowermost fibers of the inferior constrictor, otherwise known as the cricopharyngeus muscle, have their insertions in the flat portion of the cricoid cartilage and form both externally and internally the line of demarcation between the portion of the pharynx behind the larynx and the esophagus. This muscle bundle constitutes an important anatomical landmark.

FIGURE 3 The pharyngo esophageal segment (exterior view) 1 Oblique fibers of the inferior constrictor muscle 2 transverse fibers of the inferior constrictor muscle (cricopharyngeus) 3 thyroid gland 4 left recurrent laryngeal nerve showing muscular twigs 5 right recurrent laryngeal nerve enveloped by its fascia 6 esophagus 7 trachea



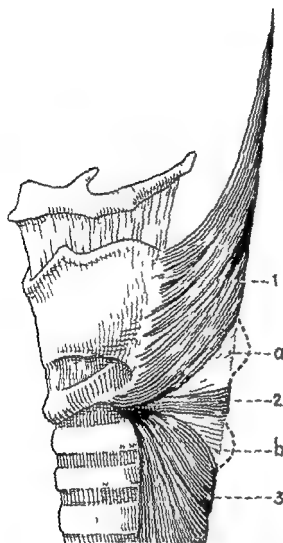


FIGURE 4 Diagram showing the weak points of the pharyngo-esophageal segment 1, Oblique fibers of the inferior constrictor, 2 cricopharyngeus muscle, 3 muscular coat of the esophagus a upper weak point b lower weak point (left lateral view)

Of equal importance is the fact that the cricopharyngeus muscle, because it acts as a sphincter plays a role of considerable importance in endoscopy as well as in the pathology of the pharyngo-esophageal segment. It is the one true sphincter of the esophagus.

Cervical Segment

The cervical portion of the esophagus extends from the tubercle of the cricoid cartilage to the level of the inferior border of the first thoracic vertebra opposite the inner ends of the clavicles. It is usually about 5 to 6 cm. in length. In front, it lies against the trachea to which it is loosely attached by strands of connective tissue and muscular fibers. Normally these two organs can be easily separated. Behind, it lies in juxtaposition to the vertebral column with the intervention of the loose cellular tissue comprising the prevertebral fascia and the underlying prevertebral muscles. On its right side, as viewed from in front, the trachea tends to cover the cervical portion and hides it almost completely. On the left side, on the contrary, the esophagus forms a flattened prominence which bulges somewhat from behind the trachea between the left lobe of the thyroid and the inferior thyroid artery. On this side the left inferior thyroid

artery and veins cross close to it. Thus the cervical esophagus is somewhat more easily exposed surgically from the left side than from the right.

The coverings of the cervical portion are part of the musculo-aponeurotic investment of the neck, the sternocleidomastoid muscles, and the superficial and middle layers of the fascia which envelops the pretracheal muscles. The carotid sheath is an obvious landmark in the surgical approach to the esophagus from either side.

On the right side the right recurrent laryngeal nerve courses against the wall of the esophagus as far as the point where it passes beneath the cricopharyngeal bundle of the inferior constrictor muscle. On the left side the recur-

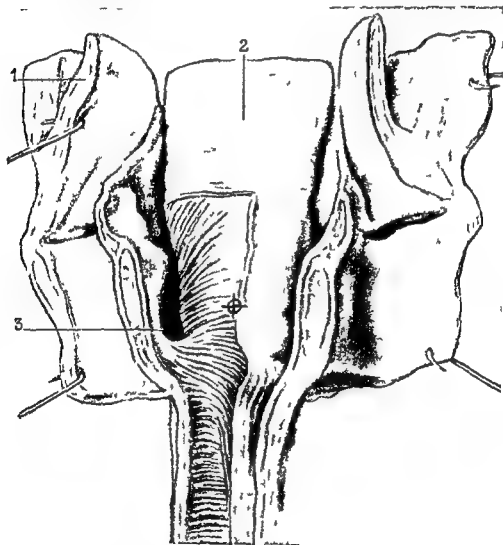


FIGURE 5 The pharyngo-esophageal segment (interior view from in front) 1 Epiglottis 2 mucosa 3 pharyngo-esophageal gutter showing the fold of Betz. The mucosa has been removed from one side to show the disposition of the muscle fibers: oblique above cricopharyngeus running transversely; esophageal fibers below. The cross and circle indicates the upper weak point. This is the place where instrumental perforations are likely to occur during esophagoscopy.

rent nerve follows the more accessible left border of the esophagus. As it enters the cervical region, this nerve lies more nearly on the anterior surface of the esophagus along the groove between the esophagus and the trachea. Thus from the surgical point of view it is important to remember that on the right side the recurrent nerve is more vulnerable and should be exposed and protected, whereas on the left the corresponding nerve lies farther away from the border of the esophagus and is less readily injured.

The cervical esophagus is completely surrounded by a layer of connective tissue which is a continuation of the peripharyngeal layer. This tissue covers the cervical esophagus completely and fuses below with the cellular tissue of the superior mediastinum. In the cervical region this layer has certain peculiarities. In front, where it is attached to the trachea, it is more dense and is made up of fibrous bands which are actually small, elastic tendinous slips mingled with more or less distinct muscular fibers. Behind, two layers of fibrous tissue, one on each side, part from the general fascial investment of the esophagus and unite posteriorly with the prevertebral fascia. They occupy a space spoken of as the retro-esophageal or retrovisceral space of Henke which is filled with loose areolar tissue and lies in continuity with the retropharyngeal space above. Because of its continuity with the superior mediastinum below, this space plays an important role in the spread of periesophageal (and retropharyngeal) suppuration and the migration of cold abscesses into the mediastinum. This layer, likewise, makes possible the mobility needed for the act of deglutition.

Thoracic Segment

This portion of the esophagus extends from the first thoracic vertebra (or in other words from the superior strait of the thorax) to the diaphragm. In length it varies between 16 and 18 cm. It contains two narrow points, one where it crosses behind the aortic arch and the other where it lies in contact with the left main bronchus. In general this portion may be subdivided into (1) the part above the bronchus and (2) the part below the bronchus.

The first or *suprabronchial portion* extends from the first thoracic vertebra to the level of the fifth thoracic vertebra. Posteriorly it lies upon the muscle layers which invest the front of the vertebral column with a bed of loose cellular tissue between. As mentioned before, this fascial layer is directly continuous with the prevertebral fascial space of the neck and it likewise provides the means for the spread of infection after perforations or in any case of periesophageal cellulitis.

In front, the esophagus in this region lies in contact from above downward with the trachea, the tracheal bifurcation, and the origin of the left main bronchus. The trachea and esophagus here are attached to one another with fibrous and fibromuscular bands which do not permit the organs to be separated except by sharp dissection. This intimate attachment favors the development of tracheo-esophageal fistulae under certain pathological circumstances.

On the right side this portion of the esophagus lies against the mediastinal pleura, and at the level of the fourth thoracic vertebra it is crossed by the azygos vein. On the left it is in apposition with the pleura, with the interposition at various points of the thoracic duct above the aortic arch, the left subclavian

artery, the left recurrent laryngeal nerve, and the aortic arch. The long contact with the left recurrent nerve accounts for the frequent occurrence, as compared with other segments, of left vocal cord paralysis in cases of carcinoma of the upper thoracic segment of the esophagus.

The aortic arch tends to compress and push the esophagus to the right, causing the constriction previously described.

The second or *sub-bronchial* portion of the thoracic segment extends from the level of the fifth thoracic vertebra to the level of the eleventh thoracic vertebra. Posteriorly, because of the interposition of such important structures as the descending aorta just above the diaphragm, the hemiazygos vein and its tributaries, the thoracic duct, the azygos vein, and the right intercostal arteries, this segment becomes separated from the vertebral column. At the lower end this separation may be as large as 3 cm.

In front it lies in contact with the tracheobronchial lymph nodes which under certain circumstances may cause pressure upon it, the left atrium, and the posterior surface of the pericardial sac. On each side are the vagus nerves, the lower periesophageal lymph nodes, the right and left mediastinal pleural reflections, and beneath these the lungs.

The topographical relations of the esophagus from the cervical region down to the esophageal hiatus of the diaphragm are illustrated in the series of cross-section drawings comprising Figures 6 to 11.

(Text continued on page 16)

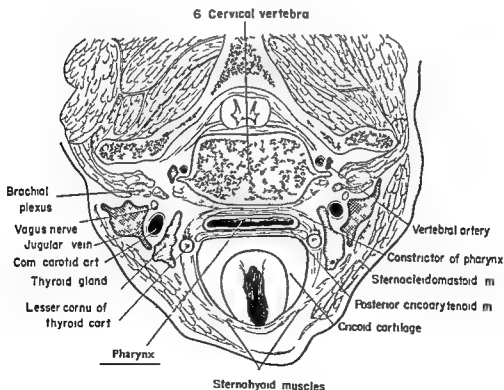


FIGURE 6 Relations of the cervical esophagus at the level of the sixth cervical vertebra (J. Delmas)

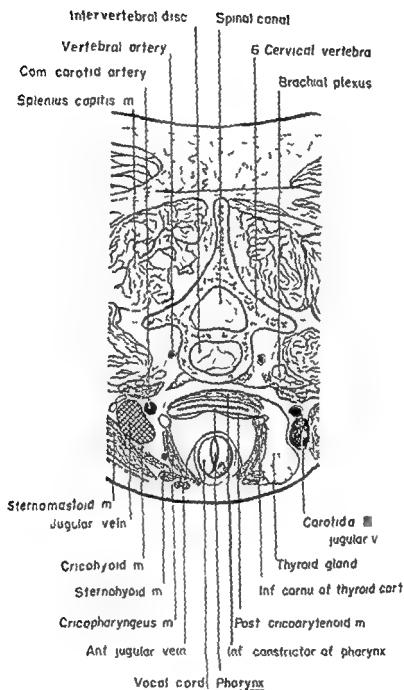


FIGURE 7 Relations of the esophagus at a level slightly lower than Figure 6 (base of the neck)
(J Delmas)



FIGURE 8 - Relations of the thoracic esophagus at the level of the second thoracic vertebra (section viewed from inferior surface) (J Delmas)

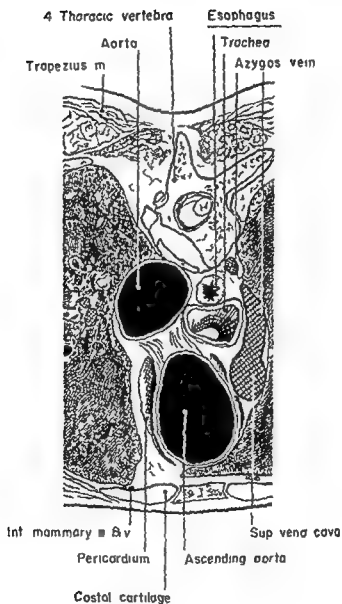


FIGURE 9 Relations of the thoracic esophagus at the level of the fourth thoracic vertebra (inferior surface) (J Delmas)

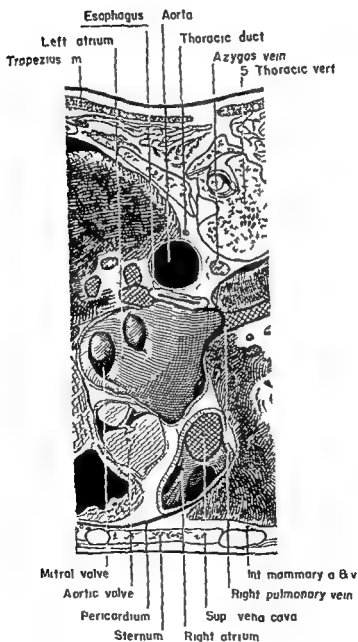


FIGURE 10 Relations of the thoracic esophagus at the level of the fifth thoracic vertebra (inferior surface) (J Delmas)



FIGURE 11 . Relations of the thoracic esophagus at the level of the seventh thoracic vertebra (inferior surface) (J Delmas)

The Diaphragmatic Segment and the Diaphragmatic Canal (Hiatus)

The esophagus becomes somewhat narrow as it enters the esophageal hiatus in the diaphragm. The hiatus is located in the right crus somewhat behind the center and at a level below the dome of the diaphragm (Fig. 12). It lies about 2 cm. in front of the vertebral column and about 1 cm. to the left of the midline of the body and usually at the level of the tenth or eleventh thoracic vertebra although in some instances it may be as high as the ninth or even the eighth thoracic vertebra. Although the length of the canal is usually stated to be no greater than 1.5 cm., in actuality it is more nearly 2 to 2.5 cm. This

disparity arises from the fact that the former figure is based upon measurements made at anatomical dissections and does not make allowance for the shrinkage of tissues under these abnormal conditions

The older anatomists, particularly Spiegel and Winslow who paid particular attention to this region, have described certain muscular fibers which pass from the diaphragm and insert into the wall of the esophagus. More recently these muscle bands were particularly emphasized by Rouget whose name they have acquired. The following description by Rouget is quoted verbatim: "I have always found in the human the rudiments of the (peri-)esophageal sphincter which is so highly developed among rodents. Quite distinct from the muscle bundles of the crura of the diaphragm which are prolonged to form the center and sides of the diaphragm, the muscle fibers which make up this structure are a little paler in color than the rest of the muscle, thinner, and not very numerous. They leave the margins of the hiatus from the inner surfaces of each pillar, pass over to the esophagus to which they are intimately attached, and end most often on its anterior surface where they form loops which decussate each with those from the opposite side (Fig. 13)."

These little, more or less well developed muscular bundles are constant in occurrence but ordinarily are found only on the infradiaphragmatic portion of the esophagus. Among the lower mammalia the presence of this muscle by its tonicity alone makes vomiting very difficult. In man, however, this muscle is too rudimentary to prevent it.

Later Juvana described two small sheets of muscle fibers, one external and

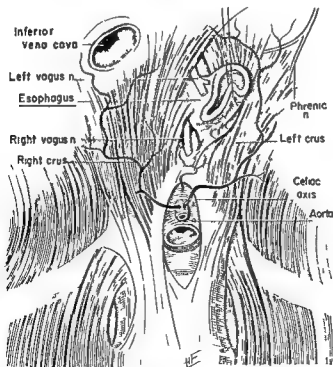


FIGURE 12 The esophageal hiatus of the diaphragm as seen from below (inferior surface)
(After Liébault)

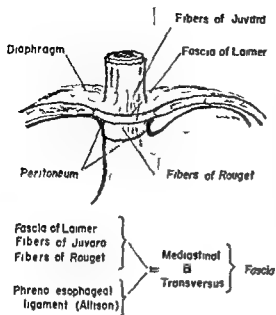


FIGURE 13 Anatomy of the esophageal hiatus. Attachments of the esophagus to the diaphragm showing the fascial investment divided at the center to reveal the layers. Note: Phreno esophageal ligament of Allison is another name for the fascia of Lamer with its attached fibers of Juvara and Rouget which actually is derived from the mediastinal fascia above and the transversalis fascia below.

the other internal which pass obliquely from the margin of the esophageal hiatus of the diaphragm to fuse with the wall of the esophagus (Fig. 13).

Still more recently a fibro-elastic sheet of tissue forming a tense circular partition extending across the hiatus from the diaphragm to the esophagus, and perforated only by two openings for the passage through it of the vagus nerves, has been described by Treitz and Lamer.

Recent investigators, on the basis of anatomical dissections, have cast doubt upon the existence of the muscular layers described by Rouget and others. It is of interest, however, that since this region has become a frequent field for the manipulations of the surgeon, as in the transthoracic repair of hiatus herniae and particularly during the operation of esophagogastricomy, it has been possible to demonstrate in many instances these muscle fibers extending from the margins of the hiatus and fusing with the muscular wall of the esophagus. The uncertainty about them arises from the fact that they are not grossly identifiable in all persons although histologically they can always be recognized.

Likewise, there is a true fascial layer between the esophagus and the diaphragm. During any dissection of the region of the lower esophagus and its hiatus through the diaphragm as seen from above, after the pleural reflection has been turned back it is obvious that the esophagus at this level is covered by a layer of fibrous tissue which disappears beneath the margins of the diaphragm. This layer arises as a thin sheet of tissue intimately attached to the esophagus at a point about 3 cm. above the level of the diaphragm. This is the level where the esophagus changes from a somewhat flattened to a truly circular form. This tissue as it descends becomes somewhat thicker and more obvious and at the superior border of the hiatus can be recognized as a definite layer. At this point it is obviously unattached to the edge of the diaphragm and although apparently adherent here to the esophagus, it can be separated from the esophageal wall easily. At the hiatus, although it still appears to be attached to the esophagus, this layer is actually a sheet of loose fibrous tissue lying beside

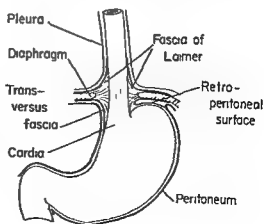
and surrounding this organ. This fact explains on the one hand the possibility of pulling the esophagus down into the abdominal cavity, to some extent, and on the other hand the ease with which the attachments of the esophagus to the diaphragmatic hiatus can be torn.

Beneath the diaphragm is a horizontal layer of similar tissue completely surrounds the hiatus, covering the anterior surfaces of the pillars which form its margins, and radiating peripherally to fuse with the retroperitoneal fascia of which it is a part (fascia transversalis). This layer is separated from the muscular fibers of the diaphragm by a layer of loose connective tissue, but it is intimately attached to the peritoneum (Fig. 15).

Thus it can be seen that the so-called fascia of Launer is actually made up of two inverted cones of tissue united by their bases (Fig. 13). The uppermost cone is actually a downward continuation of the retropleural mediastinal fascia and the lowermost cone is an extension of the transversalis fascia of the abdominal cavity. It is probably this layer which Allison calls the phreno esophageal ligament. In vertical section it delimits with the walls of the esophagus a lozenge-shaped periesophageal space filled with areolar tissue which is widest at its center and which thins out progressively at its two extremities towards the esophagus above and the cardia below. This expanse of connective tissue or fascia is reinforced by a thin layer of muscle fibers that of Juvara above and Rouget below (Figs. 13, 14, 15).

It should be stressed, however, that there is no muscular structure in this region, whether it be the diaphragm itself or the tenuous fibers of Juvara and Rouget which can play the role of a sphincter. There is absolutely no resemblance of any of these muscular structures to the cricopharyngeal sphincter mechanism the fibers of which are an integral part of the wall of the esophagus and which contract with it. In fact from the physiological point of view the diaphragmatic canal in which the esophagus lies is not a true sphincter because it does not provide intrinsic muscle fibers comparable to those at the mouth of the esophagus or at the anus. It is more nearly an extrinsic sphincter mechanism (if it has any sphincter-like action at all) similar to the relation of the levator ani muscle to the rectum.

FIGURE 14 Anatomy of the esophageal hiatus. Diaphragmatic segment of esophagus showing fascial attachments (frontal view)



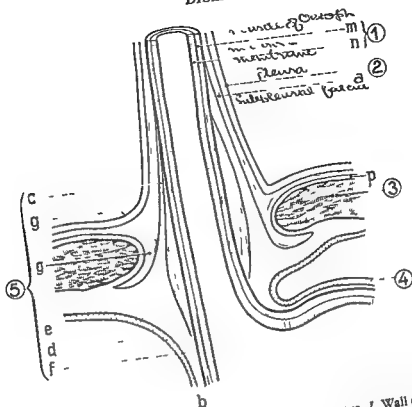


FIGURE 15 Diagram of the fascial layers at the esophageal hiatus 1, Wall of the esophagus (*m* muscularis *n* mucosa) 2 pleura (*a*, subpleural fascia) 3, diaphragm (*p*, peridiaphragmatic fascia) 4, peritoneum and retroperitoneal fascia 5, fasciae of the lower esophageal segment (*c* superior fascial layer *g* *g*, loose tissue of the sliding zone *e* subdiaphragmatic extension of inferior layer [transversalis fascia] *d* annular thickening of the fascia *f* lower extension of the fascia onto the cardia *b* retroperitoneal fascia approaching the stomach)

The Abdominal Segment

The abdominal esophagus is deeply situated posteriorly and thus difficult of access through a classical abdominal incision. It extends from the esophageal hiatus to the cardia where its termination is not always clearly defined. From the exterior the usual landmark of the cardia is the groove formed by the left margin of the lower esophagus and the fundus of the stomach. At this point a valvular mechanism comprising a fold of mucous membrane has been assumed to exist, but in fact no such valve can be demonstrated. Furthermore, there is no muscular thickening at this point and no sphincter. Likewise the folds of the mucous membrane of the lower esophagus are continuous with those of the stomach. In fact, the only distinguishing evidence of a separation between the two organs lies in the color of the mucosa. Histologically there is no transitional zone between the stratified pavement-cell type epithelium of the esophagus and the cylindrical-cell epithelium of the stomach. This abrupt change in the mucosal types actually provides the only clear-cut point of separation between the two organs. However exact the location of this point may appear to be in the cadaver, in the living subject the level of demarcation is more difficult to define. When metallic clips applied to the mucosa at the junction of the two types through the esophagoscope are viewed under the fluoroscope, wide variations in location may be observed depending chiefly on the body build of the person

In long asthenic people it lies well below the diaphragm at the first lumbar vertebra. In a short person, however, the clips may be found lying as high as the tenth thoracic vertebra. In addition, the mucosal junction as identified by these clips may be observed to change its position in the same individual the shift being indicative of the loose attachment of the mucosa to the submucosa. These changes in position are probably brought about by contraction of the *muscularis mucosae*. The upward displacement of the esophagogastric mucosal junction may actually reach as high as a point well above the diaphragm. The importance of this observation has to do with the occurrence of the so-called reflux esophagitis (Chapter 13).

After its passage through the diaphragm, the esophagus in its abdominal segment enlarges to form an inverted funnel which can be thought of as the reverse of the hypopharyngeal funnel at the proximal end of the organ. The abdominal segment lies over the tenth and eleventh thoracic vertebrae opposite the fifth left intercostal space anteriorly. In direction it veers somewhat to the left and anteriorly. It varies in length from 2 to 2.5 cm depending upon the variable factors which determine the length of any part or all of the esophagus as mentioned above. In diameter it averages 22 mm, which makes it possible to pass even the largest esophagoscopes through it. Furthermore it does not usually impede the passage of foreign bodies which have not been held up at a higher level. This is true even of rather sizable dentures.

The esophagus is in relation anteriorly with the posterior surface of the left lobe of the liver and the left vagus nerve lies upon it. Posteriorly it has no peritoneal covering but is surrounded by a bed of loose connective tissue which separates it from the left crus of the diaphragm and the aorta. To the right lies the Spiegelian lobe of the liver and to the left the fundus of the stomach. It is covered by peritoneum only in front.

The entire esophagus is enveloped by a covering of loose connective tissue containing the lymphatics, the blood vessels and the nerves, both vagus and branches of the sympathetic chain. It is this layer which permits the peristaltic motion of the esophagus and which in the diaphragmatic canal makes it possible for the esophagus to be relatively unaffected by the respiratory excursions of the diaphragm.

Blood Supply of the Esophagus

The cervical segment receives its blood supply from three sources of unequal importance. First several slender vessels, branches of the pharyngeal arteries descend upon the posterior surface of the esophagus and terminate on each side, the right more than the left. Another vessel frequently found though sometimes absent is a branch of the subclavian artery (Fig. 16). It has been called the esophageal artery of Luschka. The principal blood supply of this segment, however, is furnished by branches from the inferior thyroid arteries. They arise at various levels and are distributed to the sides of the esophagus chiefly the right. It should be pointed out that the inferior thyroid artery divides on the posterior surface of the gland to form the anterior branch which supplies chiefly the trachea but also to some extent the esophagus and the posterior branch which is distributed principally to the esophagus but in part also to the

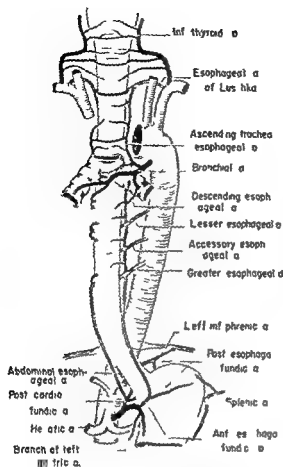


FIGURE 16 Arteries of the esophagus Type I arrangement, diagram of an anterior view (After J Calvet Coulouma and Dubas)

trachea The esophageal branches are segmentally arranged but intercommunicate from side to side and above all are united by anterior and posterior longitudinal vascular anastomoses (Figs 16 and 17)

The *thoracic segment* is supplied by a variety of vessels depending upon the level That portion which lies in the superior mediastinum above the aortic arch is supplied by vessels which descend from the neck, chiefly the terminal portion of the esophageal artery of Luschka This vessel descends principally along the left margin of the esophagus to anastomose behind the aortic arch with the anterior esophagotracheal artery described by Demel The latter arises from the posterior surface of the transverse portion of the aortic arch It is frequently supplanted by a branch of the left bronchial artery which ascends along the left side of the esophagus (Fig 16)

When the esophageal artery of Luschka is lacking, it is replaced by one of the lower branches of the inferior thyroid artery which anastomoses in the same manner and along the left side only No matter which arrangement may exist, the thoracic esophagus above the aortic arch is poorly vascularized in comparison with the cervical segment This is true especially on the right where there is no marginal anastomosis between the artery of Luschka or its counterpart from the inferior thyroid and the ascending esophageal branch of the right bronchial artery Most of the vascular intercommunications in this portion of the esophagus are within its wall

The blood supply of the *midthoracic portion*, which lies behind the aortic

arch and the left main bronchus, may present one of two common patterns although other variations are sometimes seen. In Type I the bronchial artery comprises the only source of vascularization at this level. A single branch arises from the bronchial artery and, as it reaches the left side of the esophagus, subdivides into two branches. One of these ascends along the esophagus and trachea, to both of which it gives off small branches, and anastomoses with the artery of Luschka or its counterpart just above the aortic arch. The other subdivision, which supplies the esophagus only, descends to just below the bronchial bifurcation where it joins the first aortic esophageal artery. The left bronchial artery still further along its course gives off another smaller vessel which anastomoses with the preceding branch. The right bronchial artery, like the left, gives off an ascending esophagotracheal branch and one which descends along the esophagus. But these vessels are of less importance than those from the left side and do not anastomose with any other vessels either above or below (Fig. 16).

In Type II the ascending esophagotracheal and the descending esophageal branches described above come off directly from the inferior surface of the aortic arch, the first from near the termination of the arch and the second from the point where the arch ends in the descending aorta (artery of Gossart).

Whichever arrangement may prevail the blood supply of the midthoracic esophagus is actually arranged in two levels. The superior (aortotracheal) level is composed of the right and left ascending esophagotracheal vessels, which by their anterior and posterior anastomoses surround the esophagus and in some

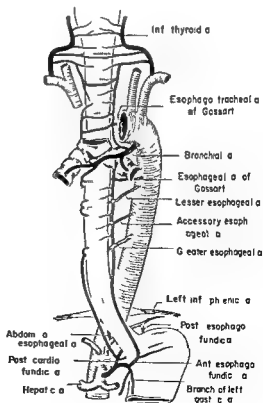


FIGURE 17. Arteries of the esophagus. Type II arrangement. Diagram of anterior view. (After J. Calvet, Coulouma and Dubas.)

cases are joined on the left by the anterior esophagotracheal branch. The inferior (or intertracheobronchial) level comprises the right and left descending esophageal arteries, which are united behind the esophagus by one or two anastomoses so that at this point the esophagus is encircled by an arterial ring made up in front of the bronchial arteries and behind of the above-mentioned anastomoses. Thus, it is obvious that the midthoracic segment is richly vascularized.

The lower portion of the thoracic esophagus, from the level of the left main bronchus down, is usually supplied only by branches direct from the descending aorta, with branches from the intercostal arteries in exceptional cases. In general, these aortic esophageal branches number three or four, but there may be only one (Figs 16 and 17).

The lower thoracic esophagus, from the point of view of its vascularization, may be divided into two segments of unequal importance. The upper begins just below the bronchial arterial system, or the descending esophageal artery when it exists, and depends for its blood supply upon the lesser esophageal and the accessory esophageal arteries. These two rather slim vessels are united along the left side of the esophagus by a fine anastomotic arcade which is in continuity above with the descending esophageal branch of the bronchial artery or the descending esophageal vessel from the aortic arch, whichever exists, and below with the ascending branch of the greater esophageal artery. Each has a retro-esophageal branch which vanishes before reaching the right side. In addition, thin pre-esophageal and retro-esophageal branches extend from this above-mentioned tenuous longitudinal arcade toward the right side of the organ but it should be emphasized that these vessels, even when they actually reach the right side of the esophagus, which is not always the case, never enter into a marginal anastomotic arcade like that along the left side.

The lower segment of this portion of the esophagus begins at the origin of the greater esophageal artery, the caliber of which, along with that of most of its branches, is of considerable size. This artery, the largest coming directly from the aorta, ordinarily gives off three branches: one anterior, a second posterior, and the third a branch which passes crosswise to the right side of the esophagus. Branches of all three of these subdivisions anastomose within the esophageal hiatus of the diaphragm with the anterior and posterior ascending terminal branches of the left gastric artery. They all give rise to numerous twigs forming a network which insures a rich vascularization of the lower thoracic esophagus (Figs 16 and 17).

The portion of the esophagus which traverses the hiatus through the diaphragm is supplied by the above-mentioned anastomotic communications with the greater esophageal artery, and above all by a rather constant branch of the left inferior phrenic artery which joins the posterior branch of the greater esophageal artery.

The abdominal segment of the esophagus is supplied by anterior and posterior cardiofundic arteries which arise from the arcade of the left gastric artery. In addition, the left inferior phrenic artery often furnishes a posterior esophagocardiofundic branch, and sometimes the splenic artery gives rise to a similar vessel. Another branch arises from the superior suprarenal artery. All these branches which anastomose more or less directly with the branches of the



FIGURE 18 Example of the segmental nature of the esophageal blood supply. Lateral view with partial filling of segment below the level of the aorta and the azygos vein. Film shows the esophageal arteries proper and the submucosal network which is particularly distinct above the diaphragm at the lower portion of the film. (After J. Calvet and Poulhès.)

greater esophageal artery bring about an ample vascularization of the abdominal esophagus.

In general, it should be emphasized that the blood supply of the esophagus by way of the branches already described tends to be segmental in distribution (Fig. 18). This fact is important to the surgeon who, in order to prevent failures of blood supply, must avoid extensive interruptions of these vessels beyond the limits of the level at which the esophagus is to be cut across in the performance of an esophagectomy.

The Venous Drainage of the Esophagus (Fig. 19)

Blood coming from the cervical and superior mediastinal segments of the esophagus is diverted by small venous channels into the innominate veins and superior vena cava by way of the inferior thyroid, the bronchial, and highest intercostal veins. Lower down in the midthoracic segment the drainage is into

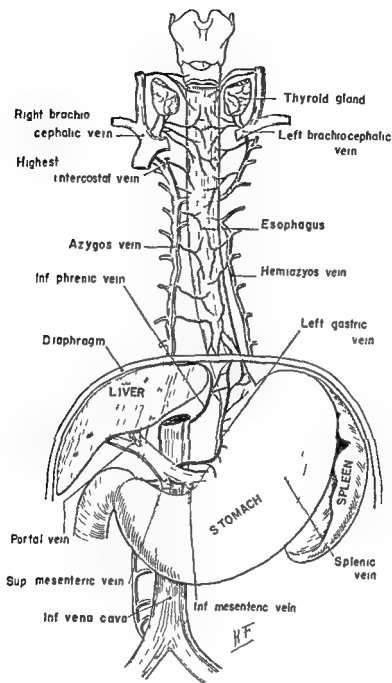


FIGURE 19 The venous circulation of the esophagus

the azygos and hemiazygos veins and thus into the superior vena cava. From the lower third of the organ, however, the blood enters the portal system by way of the branches of the left gastric veins and the upper or paracardial branches and vasa brevia of the splenic vein. Some of the blood from this region passes off through the left inferior phrenic vein.

Throughout the entire length of the esophagus there is an important submucosal plexus of veins. There is also a periesophageal plexus which, though scanty above, is very well developed in the lower third. These two plexuses are joined by multiple anastomotic channels which penetrate the muscular coat of

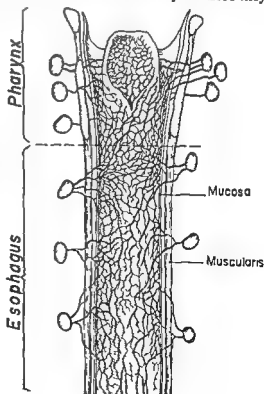
the esophagus. As is well known, these venous channels become enormously enlarged in portal hypertension to form the varices which are so often the source of serious hemorrhages in cirrhosis of the liver or thrombosis of the splenic vein (Fig. 19).

The Lymphatic Drainage of the Esophagus

The lymphatic channels of the esophagus arise as two groups, one from the mucous membrane and the other from the muscular layer. The numerous lymphatic capillaries of the esophagus are arranged longitudinally so as to extend the entire length of the organ, uniting with those in the pharynx above and the gastric mucosal channels below. This network is particularly thick in the deep layer of the mucous membrane and in the submucosa. The network of capillaries in the muscular layer is in part independent and in part connected with that of the mucosal layer (Fig. 20).

The collecting trunks which arise in the submucosal layer follow a course which varies according to their origin. Some traverse the muscular layer and end in the nearest adjacent lymph nodes, others run beneath the mucosa but follow a characteristic pattern. The direction of flow in the vessels of the upper two thirds tends to be upward whereas in those of the lower one-third it is downward. The frequency with which downward metastases coming from carcinomata of the midthoracic segment are observed indicates that there is certainly a descending flow within the collecting trunks of this area as well. All of these channels course long distances within the submucosal layer before they

FIGURE 20 The lymphatics of the esophagus. Diagram shows the intricacy of the lymphatic vessels, their wandering course extending long distances, and their tendency to overlap from one region to another. (After Rouviere.)



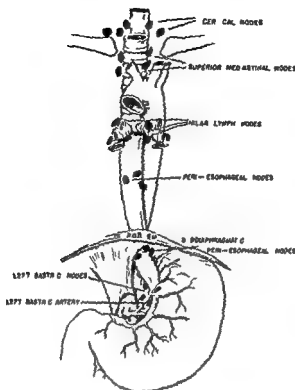


FIGURE 21 Lymph node groups which drain the esophagus at various levels

traverse the muscularis to reach their termination. The trunks which come from the capillary networks of the mucosa and the muscularis do likewise but to a lesser extent.

Once they have pierced the muscular layer, all of the collecting trunks pass directly to the nearest lymph nodes. The nodes which are principally concerned are from above downward, those along the internal jugular veins in the neck, the paratracheal nodes in the superior mediastinum, the peribronchial and subcarinal nodes in the midthoracic region, the periesophageal nodes in the lower mediastinum, the nodes in the pulmonary ligament, and, below the diaphragm, the paracardial group and those in the region of the branches of the left gastric artery and vein along the upper portion of the lesser curvature of the stomach (Fig. 21).

In general, because of the arrangement of the lymphatic trunks, the nodal metastases tend to involve those groups of nodes nearest the segment in which the growth is located. It should be pointed out, however, that although this is the usual occurrence, there are innumerable instances where because of the rich anastomoses of the lymphatic channels metastases may be discovered in lymph nodes which lie a great distance from the primary site. Thus cervical or subdiaphragmatic metastases, either or both, may be seen coming from a carcinoma in the midthoracic segment, and superior mediastinal paratracheal nodal involvement is commonly observed in carcinoma of the cervical segment. Furthermore, because of the rich anastomoses of the intramural channels, carcinomata of the cardiac end of the stomach frequently metastasize to the lower mediastinal nodes.

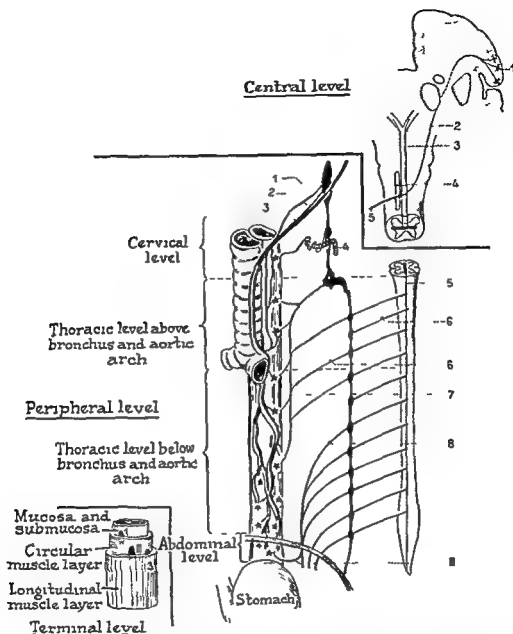


FIGURE 22 Innervation of the esophagus (Terracol and Y Guerrier)

Central (cerebral) level 1 nerve cells at the foot of the anterior central gyrus 2 geniculate bundle 3 intra axial sympathetic pathways in communication with the vegetative nuclei of the floor of the third ventricle 4 nucleus of the vagus nerve 5 vagus nerve

Peripheral nerve level 1 2 vagosympathetic anastomoses 3 vagus nerve 4 inferior thyroid artery and its sympathetic nerve plexus 5 intermedullary tract (vegetative medullary centers) 6 communicating white rami and the posterior mediastinal splanchnics 7 lateral esophageal collateral sympathetic trunk 8 thoraco-abdominal splanchnics 9 esophageal branch of the great splanchnic trunk

Terminal level The differing positions of the Auerbach plexus in the tunics of the esophagus 1 in the submucosa 2 between the two muscular layers 3 in the outer muscle layer

DISEASES OF THE ESOPHAGUS

The Innervation of the Esophagus

The innervation of the esophagus poses some complex problems. An intramural nervous system having rich intercommunicating anastomoses receives its impulses from two antagonistic trunks, namely the vagus and the sympathetic (Fig 22)

The Extrinsic Nervous System of the Esophagus

The *pharyngo-esophageal segment* is usually innervated by a plexus derived from the branches of the superior laryngeal nerve, the cervical sympathetic trunk, and particularly the external laryngeal nerve (Fig 23). But this plexus is not constant. The mouth of the esophagus is the most important region in this area. Here in the *pars fundiformis* of the hypopharynx the principal innervation is supplied by the recurrent laryngeal nerve from which numerous thin branches are distributed to the esophagus without crossing the midline and without actually forming a plexus. This disposition of the fibers explains certain functional disorders of the cricopharyngeal punchcock mechanism. The importance of these nerve fibers in activating a complicated sphincter-like behavior of the muscles at this point is consistent with the fact that the muscle fibers at the mouth of the esophagus and in its upper one-third are striated and the fact that the recurrent nerve is the only available source in this region of cerebrospinal peripheral innervation to supply them. By contrast, the lower portion of the esophagus, whose muscular tunic is composed of smooth muscle fibers is innervated only by sympathetic and parasympathetic fibers.

The *cervical esophagus* receives its innervation from the recurrent laryngeal nerves and from the cervical sympathetic plexus (Fig 23). The vagal innervation is by way of small filaments which arise from the recurrent nerves. The left recurrent nerve lies in the groove between the esophagus and the trachea. The

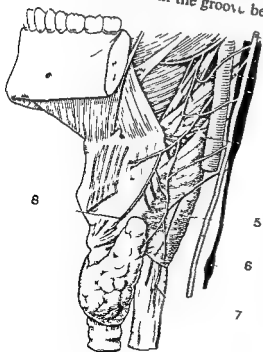


FIGURE 23 The plexus of Haller and its esophageal branches. 1 Vagus nerve 2 superior laryngeal nerve 3 superior cervical sympathetic ganglion 4 sympathetic root of the superior laryngeal nerve 5 inferior constrictor muscle 6 middle cervical sympathetic ganglion 7 esophageal nerves 8 external laryngeal nerve. Note The pharyngo-esophageal segment is innervated by branches known as esophageal nerves which arise from a plexus formed by branches of the external laryngeal nerve the vagus nerve and the cervical sympathetic nerves (After P Couloumas and Van Varseveld)

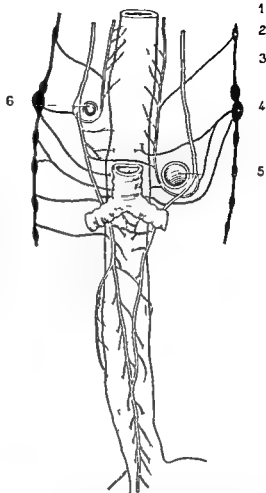


FIGURE 24 Innervation of the esophagus by the vagus nerves 1 Recurrent nerve 2 middle cervical sympathetic ganglion 3 vagus nerve 4 inferior cervical sympathetic ganglion 5 aorta 6 right subclavian artery *Note* This diagram shows the anastomoses of the sympathetic and the recurrent nerves the vagosympathetic plexus behind the trachea and bronchi the direct fibers of the thoracic sympathetic supplying the esophagus above and below the bronchus on the right and above the bronchus on the left (After P. Couloumas and Van Varsveld)

right follows the free border of the trachea on that side. Because of its origin below the aortic arch the former is the chief source of branches to the superior mediastinal portion of the esophagus by way of a branch sometimes called the great lateral nerve of the esophagus, which emerges from the main trunk as it winds beneath the aortic arch. A similar lateral esophageal branch comes off the right recurrent nerve as it winds under the right subclavian artery. The terminal branches of these nerves do not cross the midline and do not form a plexus (Fig. 24).

The sympathetic innervation of the cervical segment comes from the laryngeal plexus and the plexus around the inferior thyroid artery which may be formed by filaments from the superior cervical ganglion, from the cervical sympathetic chain or from the superior cardiac branch of the vagus nerve. On the right side the inferior cervical ganglion may give rise to a single fiber (Fig. 24). On the left Delmas and Laux have described a long nerve arising from the stellate ganglion which sends several filaments to the superior mediastinal segment (Fig. 25).

The sources of all the nervous elements of this region, however, are difficult to recognize, and in spite of the investigations of some of the greatest anatomists

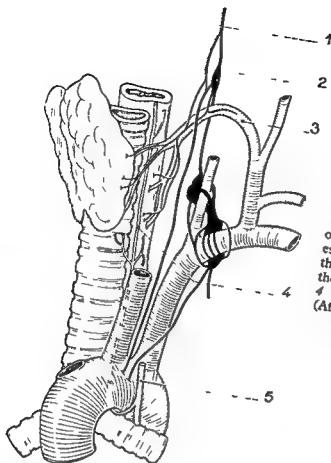


FIGURE 25 The sympathetic plexus of the inferior thyroid artery and its esophageal branches 1 Cervical sympathetic trunk 2 middle cervical sympathetic ganglion 3, inferior thyroid artery 4 recurrent nerve 5 left vagus nerve (After P. Couloumas and Van Varsveld)

it is difficult to arrive at definite conclusions about them. Although the innervation of this portion of the esophagus is exceedingly variable,* it arises chiefly from nerve trunks destined to supply the larynx.

* Recently A. Flottes of Montpellier has reported the results of his dissection of the nerves of this region in ten subjects. In three instances he was able to demonstrate a plexiform network of branches emanating from the recurrent nerves and from the periarterial filaments. The latter, consisting of thin fibers, approached the esophagus at a point about 2 cm. below the inferior pole of the thyroid gland and passed horizontally toward the esophageal wall, crossing the vagal fibers with which they became involved in an intricate anastomosis before penetrating the superficial muscle layer.

In a fourth case a juxta articular plexus formed from the terminal branches of the recurrent nerve and two branches of the superior laryngeal nerve was discovered behind the cricoarytenoid articulations. Three branches emanated from this plexus, descending laterally along a distance of 1 or 2 cm. of the anterior surface and sides of the esophagus.

In six other dissections the nerves of the cervical esophagus came entirely from the vagus arising

Once by two branches from the lateral cricopharyngeal nerve
Once by a bifurcation situated below the cricoid ring (two esophageal branches with a horizontal direction and one branch continuing in the usual direction from the inferior laryngeal nerve)

Once a branch emerging from the loop of Galen followed a recurrent course 3 or 4 cm. along the sides of the esophagus
Once by two parallel branches separated by a 5 cm. interval which anastomosed in the midline with their homologues from the opposite side

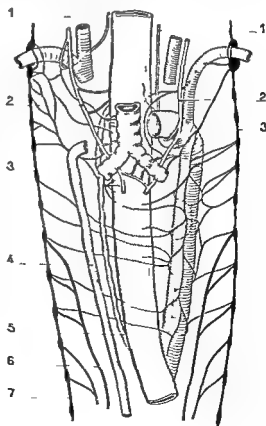
Once by a single branch coming off the recurrent nerve in front of the posterior fibers of the cricoarytenoid muscle
And finally once by a branch having a common trunk with a twig going to the cricothyroid articulation

The innervation of the *thoracic segment* of the esophagus The vagal innervation of the superior mediastinal portion varies from one side to the other *On the right* branches supplying the anterior surface of the esophagus emerge from four or five tracheal branches which arise in turn from the right vagus trunk. In addition three or four small filaments leave the right recurrent laryngeal nerve directly to supply its lateral surface (Fig. 24) *On the left* this portion is innervated directly by segmental branches of the left recurrent nerve or indirectly by branches from the left great lateral nerve of the esophagus which is very inconstant (Fig. 24)

The sympathetic innervation of the superior mediastinal segment depends upon branches from the inferior cervical ganglion and to a variable extent from the first thoracic ganglion *On the right side* the inferior cervical ganglion furnishes one strand, and three or four other fibers of thoracic origin pass behind the vagus nerve and beside the thoracic duct to the esophagus *On the left* this segment receives a long branch arising from the superior portion of the inferior cervical ganglion or from the aortic plexus (Fig. 26)

Behind the bifurcation of the trachea each of the vagus nerves is subdivided into two or three bundles held together by a fascial sheath and communicates with the corresponding nerve of the opposite side by anastomotic transverse branches which make up the pulmonary plexus This plexus anastomoses in turn with the thoracic sympathetic chain which participates in the innervation of the esophagus at this point by way of the aortic plexus which is in turn made

FIGURE 26 The innervation of the esophagus by the thoracic sympathetic. 1 Recurrent nerve 2 vagus nerve 3 thoracic sympathetic chain 4 azygos vein 5 thoracic duct 6 greater splanchnic nerve 7 lesser splanchnic nerve *Note* The direct fibers for the esophagus above and below the bronchus are on the right and for the esophagus above the bronchus are on the left The innervation of the esophagus below the bronchus on the left comes from the aortic plexus and the vagosympathetic plexus behind the trachea and bronchus



up of filaments from the second to the seventh thoracic ganglia. The aortic plexus supplies the majority of the esophageal branches which approach the esophagus from in back or penetrate its wall along with the esophageal arteries of aortic origin. The thoracic sympathetic trunk also sends branches directly to the esophagus from the thoracic ganglia and from the great splanchnic nerves. These enter it along with the branches of the azygos and hemiazygos veins (Fig. 26).

In the portion of the esophagus between the bronchus and the diaphragm the vagal innervation is maintained at all levels by branches of the main vagal trunks, which in the lower portion of this segment change their relation to the esophagus so that the left nerve lies in front and the right nerve behind the organ. One tributary branch has been singled out by Van Varseveld under the name of 'the great posterolateral esophageal nerve of the vagus'. It leaves the right vagus nerve at a point just above the bronchus and is distributed all along the posterior surface of the esophagus.

The sympathetic innervation to this portion of the esophagus on the right side is by means of filaments arising directly from the third and fourth thoracic ganglia and passing behind the thoracic duct and the azygos vein. On the left the innervation is provided above by branches of the aortic plexus which pass behind the hemiazygos vein and further down by filaments arising from the greater splanchnic nerve (Fig. 26).

The innervation of the lower esophageal segment is particularly interesting because of the varying patterns of distribution which may be encountered. In general, it may be said that the vagus nerves are the only source of sensory and motor innervation of this portion of the esophagus, and that the sympathetic by way of its perivascular branches probably has merely a vasomotor function.

In the arrangement usually described, the vagus nerves as they leave the subbronchial region subdivide into parallel longitudinal branches. Those from the right nerve cover the posterior surface of the esophagus while the branches of the left cover the anterior aspect. As they descend, the branches fan out into abundant anastomosing filaments to form around the lower portion of this segment of the organ the periesophageal plexus. The majority of these plexiform fibers reunite posteriorly to form the abdominal vagus nerve. This nerve is usually considered to be the reconstituted right nerve which, without giving off any important branches to the abdominal esophagus or cardia, passes to the semilunar ganglion and the tributary vascular plexuses around the celiac axis and thence to most of the abdominal viscera as far down as the rectum. On the anterior surface of the lower esophagus some of the collateral branches of the periesophageal plexus pass as several trunks or sometimes as a single nerve, usually thought of as the termination of the left vagus, to the anterior surface of the stomach below the cardia where it vanishes in the gastric wall. It is this trunk which supplies most of the branches to the abdominal segment of the esophagus (Fig. 27).

Because of the revival in recent years of the surgical interruption of the vagus nerves for the treatment of certain disorders of the gastrointestinal tract particularly ulcer of the duodenum, there has been a renewal of interest in the careful study of the anatomical pattern of these nerves. As a result it has been

shown by various authors, both anatomists and surgeons, that there are many variations from the usually described pattern. In order to overcome the confusion which inevitably results from so many conflicting reports and variable descriptions, it may be useful to present a new concept of the vagus nerves as elaborated by J Delmas. This presentation, because of its logic and simplicity as well as its conformity with our knowledge of the embryological development of the organs involved, imparts both an anatomical and a functional significance to the facts observed.

It has been recognized for some time that fibers emerging from the periesophageal plexus become intermingled to such an extent that the emergent trunks usually single but in some cases multiple, are made up of mixtures of fibers from both right and left nerves of origin. The large posterior trunk, usually referred to in the past as the right but today often called the chief abdominal vagus trunk, thus is formed from a confluence of elements from both right and left nerves, consistent with the fact that in the abdominal cavity the viscera of the alimentary canal being unpaired and actually median in location and origin can have but one nerve pedicle.

The abdominal vagus (the reconstituted right nerve) comprises almost exclusively all the parasympathetic innervation of the celiac plexus. Further down by the intramesenteric branches it proceeds by way of the superior and inferior mesenteric plexuses to supply the innervation for the entire alimentary canal just as the aorta by its unpaired medial branches provides the circulation for the entire digestive tract including the rectal ampulla. Furthermore the much smaller anterior vagal branch known classically as the reconstituted left vagus nerve is actually nothing but the first abdominal visceral branch whose characteristic distribution is to the submesocolic viscera namely the liver the biliary passages, and the anterior surface of the stomach. These are the structures which because of their development between the two leaves of the anterior mesogastrium of the embryo, would require a special pedicle which can be established only after the esophagus emerges from below the diaphragm.

Thus the two vagus nerves which are quite distinct from their point of origin to the upper limit of the lower esophageal segment as it emerges from beneath the left main bronchus become intermingled as they divide to form the periesophageal plexus from which finally a single terminal abdominal vagal trunk emerges. The point at which this occurs corresponds to the level below which there are no more paired or bilateral viscera of endodermic origin (lungs thyroid etc). The anterior trunk (left vagus of the classical descriptions) is actually a collateral branch of the plexus which innervates all the viscera developed within the anterior mesogastrium. The abdominal vagus (or posterior trunk) is destined to innervate all the digestive tract the rectum included.

One may deduce from this that there is practically speaking an equal admixture of the fibers from the right and left nerve trunks which takes place in the periesophageal plexus and which is certainly the anatomical reason for the formation of this plexus. As one could logically foresee the digestive tract including the lower thoracic and abdominal segments of the esophagus possesses merely a single unpaired median nerve trunk.

This concept helps also to understand the double innervation of the esoph

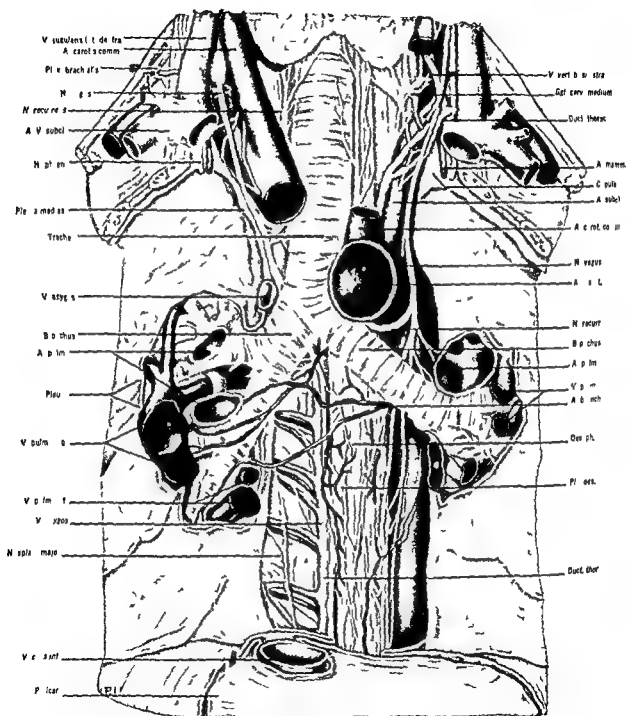


FIGURE 27 Course of the vagus nerve (From Tondury, *Angewandte und topographische Anatomie*, Georg Thieme Verlag Stuttgart 1951)

agus The cervical segment whose muscle fibers are striated receives cerebro-spinal nerve filaments by way of the eleventh cranial nerves (spinal accessory) whose collateral branches run with the recurrent laryngeal nerves The lower three fourths of the esophagus, however, the muscle fibers of which are non-striated is innervated by the vagus nerves

The Intrinsic Nervous System of the Esophagus

The plexuses of Meissner and Auerbach are most highly developed in the lower one third of the esophagus The various constituent elements of these plexuses, consisting of nerve filaments and ganglion cells, are principally distributed in the very narrow layer of connective and vascular tissue which separates the two layers of smooth muscle in the esophageal wall A certain number of these elements may also be encountered within the substance of the muscles themselves A microscopic field of a given size and thickness in the lower third of the organ may contain as many as five or six groups of ganglion cells and nerve filaments, whereas a similar field from the middle or upper third may contain only two or three Furthermore, each group of ganglion cells is more rich in cellular and nervous elements in the lower third than at the upper levels However, at the lowermost portion of the inferior third just proximal to the cardia, the number and importance of these structures decrease rapidly

The cells of the plexuses are multipolar with five to twelve very fine dendrits which, by means of intercommunications with each other, form an uninterrupted continuous network between the muscle layers The cellular elements are in continuity with each other so that nerve tissue actually extends without interruption from one cell to the neighboring cells thus producing a sheet of nerve tissue between the muscle layers The network is much more dense and its constituent elements much finer the nearer it lies to the esophageal mucous membrane layer This fibrillar network comprises the basic element of the intrinsic nervous system of the esophagus and of the entire alimentary canal It receives communicating fibers from the vagus nerves It has connections with the sensory fibers of the mucosal layer and it sends numerous filaments to the muscle layers to bring about the tonicity and contractility of the organ

The vagus nerves supply both myelinated and unmyelinated fibers to the esophagus The former all terminate in the plexuses of Auerbach and Meissner The majority of the unmyelinated fibers make up a nerve network having large meshes which surround the cells of the intramural plexuses This network communicates with the nerve cells but remains clearly differentiated from the intrinsic plexuses The fibers of this meshwork terminate as fibrillar plaques which surround the plexus cells

Other unmyelinated fibers from the vagus nerve pass directly to the muscle fibers and end in contact with the nuclei of these cells Certain of these nerve fibers traverse the intramural plexuses without giving off any communicating fibrils and end in the subjacent muscular cells of the muscularis mucosae These motor fibers are important because they insure the only motor innervation when certain lesions of the esophageal wall have altered the character of the intrinsic intramural nervous system

Whether myelinated or not the vagal fibers which communicate with the

plexuses of Meissner and Auerbach come from cells contained in the vagal nerve ganglia, chiefly the plexiform ganglion, or even from the main trunk of the nerve

INTRINSIC SENSORY INNERVATION The sensory innervation of the esophagus has to do with two types of intramucosal nerve elements. One, consisting of encapsulated endings identical with the tactile bodies of the skin, is found in the mucosa throughout the entire length of the organ. The other consists of oval-shaped bodies which lie unattached beneath the epithelial layer. All these sensory corpuscles consist of leaf-shaped, flattened terminations arising from very thin and widely branching filaments. They form an almost complete network within the mucosal layer. They are surrounded by an arborization of nerve fibers, the cell bodies of which are a part of the plexus of Meissner situated in the submucosa. Other myelinated fibers arise from Meissner's plexus and pass directly to the smooth muscle fibers.

Thus there is within the wall of the esophagus a truly autonomic nervous system with its own intramural reflex arc. Recognition of this fact makes it possible to understand certain aspects of the motor dysfunctions of the esophagus and of the entire digestive tract as well.

PART II MICROSCOPIC ANATOMY

The esophagus has three principal layers, the mucous membrane, the submucosal layer, and the muscular layer (Fig. 28). Of great importance to the surgeon is the fact that it does not possess a serosal layer.

The *mucous membrane* consists of several layers comprising the epithelium, the chorion or tunica propria, the muscularis mucosae, and the glands.

The *epithelium* of the esophagus varies depending on its location in relation to the diaphragm. In the long segment above the diaphragm the epithelium is of the stratified pavement-cell type without a cornified layer (Fig. 29). It is similar to that of the buccal mucosa. In the embryo it is simple at first, but at the third month it becomes stratified and covered with cilia. The cells then undergo changes in shape to the pavement type and finally lose their cilia.

In the portion below the diaphragm the epithelial covering undergoes an abrupt change in its structure at a point just below the level of the esophageal hiatus. Here it changes to a cylindrical-cell type like that found in the stomach. It consists of numerous mucus-containing cylindrical cells (goblet cells) and of numerous glands which often encroach upon the chorion.

The epithelial layer rests upon the *chorion* or *tunica propria*, a dense papillary layer which along the entire length of the esophagus is normally heaped up in longitudinal folds which give the mucosal layer the appearance of being arranged in festoons when viewed in cross section (Fig. 28). This layer is made up of connective tissue fibers and elastic fibrils, the latter being grouped chiefly in a longitudinal direction. The chorion layer is richly supplied with lymphoid tissue which consists of closed follicles surrounding the excretory ducts of the

glands. However, in the lower part of the esophagus this tissue is more or less diffuse.

The *muscularis mucosae* consists of both longitudinal and circular smooth muscle fibers (Fig. 30). It begins at the junction of the esophagus with the pharynx. Nearest the tunica propria it is composed of isolated small bundles of smooth muscle fibers arranged longitudinally. At a deeper level it becomes a continuous layer extending the entire length of the esophagus between the mucosa and the submucosa.

The *esophageal glands* are of two types. Those which are characteristic of the esophagus itself are tubular in form and sparsely distributed throughout the submucosa. They are most numerous along the lateral and posterior portions of the circumference of the organ. Their secretory cells are partly serous but chiefly mucus producing. Their ducts are usually surrounded by lymphoid tissue in the mucosal or submucosal layers. In the distal portion of the esophagus the glands are similar to those of the cardiac end of the stomach. These are true mucous glands of the tubular or acinous type, some of which have the structure of those found in the fundus of the stomach. A curious fact is that occasionally

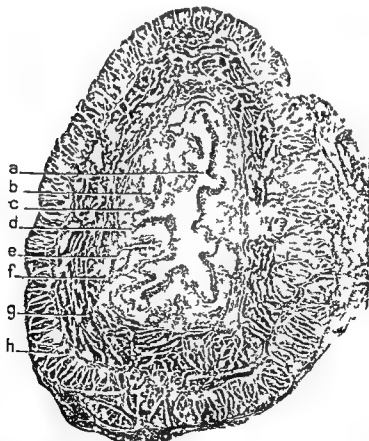


FIGURE 28 Cross section of the normal esophagus (adult man) *a* Lining epithelium *b* festooned contours of the lumen *c* chorion *d* muscularis mucosae *e* glands *f* submucosa *g* circular muscle fiber layer *h* longitudinal muscle layer $\times 130$



FIGURE 29 Normal esophagus (adult man) showing *a* stratified pavement type epithelial lining *b* vascular connective tissue of the chorion $\times 130$



FIGURE 30 Normal esophagus (adult man) showing mucosa and submucosa *a* Epithelium *b* chorion *c* muscularis mucosae *d* glands, *e*, submucosa *f* circular muscular layer $\times 50$

glands of this type may occur in the upper end of the esophagus from the level of the cricoid to a point opposite the fifth tracheal ring

The submucosal layer which is made up of loose, mobile, elastic connective tissue contains the trunks of the vessels and nerves as they proceed to the mucosa. Its loose attachment permits changes of position of these structures in relation to the muscular layers. It is not at all unusual to observe islands of glandular tissue within this layer (Fig 30), and in the upper third of the esophagus the submucosa may contain ectopic islands of gastric mucosa hardly any larger than the head of a pin or a grain of rice. These are structural anomalies representing vestigial remnants of the primordial endodermal digestive canal. They may play a role in the formation of cysts and may be responsible for the development of esophagitis and ulcerations in this segment of the organ.

The muscular layer in the lower pharynx and upper one-fourth of the

FIGURE 31 Normal esophagus (adult man)—longitudinal section of the posterior wall *a* epithelium *b* chorion *c* muscularis mucosae *d* submucosa *e* circular muscle layer consisting entirely of smooth muscle fibers as is also *g* the longitudinal muscle layer *f* ganglion cells of the intermuscular sympathetic network $\times 35$

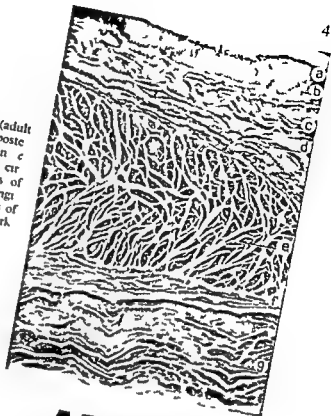


FIGURE 32 Normal esophagus Upper third of the section is at the hiatus lower two-thirds is below the diaphragm (six months infant—longitudinal section) *a* Fold of esophageal mucosa covered with pavement epithelium at *b* and with cylindrical epithelium at *c* *d* muscularis mucosae *e* submucosa *f* circular smooth muscle fibers *g* longitudinal smooth muscle fibers *h* circular striated muscle bundles of the diaphragm *i* circular striated muscle fibers emanating from the diaphragm and inserting into the adventitia of this portion of the esophagus and constituting the external muscle of the lower esophagus *j* and *k* which is never commingled with the longitudinal smooth muscle layer *g* from which it is separated by a connective tissue septum which is visible on this photomicrograph *l* subdiaphragmatic periesophageal lymph node *m* neurovascular bundle $\times 10$



esophagus consists of bundles of striated fibers intermingled with bundles of smooth muscle fibers. The relative number of smooth muscle fibers increases gradually from above downward and in the lower third smooth muscle fibers are the only type found. A few bundles of striated fibers may be found close to the cardia, but they do not constitute a sphincter. The muscle fibers are arranged in two layers, an inner circular layer and an outer longitudinal layer separated by a layer of vascular connective tissue containing the network of nerve fibers and ganglion cells which makes up the intrinsic nervous system of the esophagus (Figs. 31 and 32). The muscular coat as a whole varies in thickness from the diaphragmatic region, where it is relatively thick, to the cervical segment, just below the pharynx, where it is particularly thin. The inner layer of circular fibers is thinnest at the beginning of the esophagus but increases in thickness progressively from above down to the diaphragmatic area where it is most thick. The longitudinal outer layer is thin in the portion of the esophagus behind the bifurcation of the trachea and is relatively thick at each extremity.

CHAPTER 2

Physiology

THE ESOPHAGUS is essentially an organ of transit. Within it takes place the last phase of deglutition but its role is by no means a passive one. Several factors should be considered in the study of the physiology of deglutition: first, the nature of the material swallowed (whether liquid or solid); secondly, the behavior of the several parts of the esophagus as for example the narrow segments and particularly the longer uncontracted segments; and finally, the effect of position of the subject whether erect or lying down.

The Phases of Deglutition

The function of the *mouth of the esophagus* is bound up with the mechanism referred to by Chevalier Jackson as the cricopharyngeal pinchcock. The principal participant in this mechanism is a particular bundle of the lowermost fibers of the inferior constrictor of the pharynx. Normally in the resting state, the mouth of the esophagus remains closed by a tonic contraction which serves to prevent the ingestion of air at each inspiration. During the act of swallowing however the cricopharyngeal muscle bundle relaxes. This relaxation is normally timed to coincide with the moment of contraction of the inferior constrictor muscle. Usually this opening mechanism is a phenomenon of reflex origin arising in the pharynx but it can be otherwise. In fact the mouth of the esophagus can on occasion be held open at will.

When the larynx is closed the bolus of food slides over each side of the epiglottis into the pharyngolaryngeal gutters where propelled by the muscles of the pharynx it engages within the mouth of the esophagus which in turn opens by the reflex action mentioned above whereupon by a sort of rhythmic motion it descends into the esophagus. Perfect coordination must take place between the pharyngeal propulsion impulse and the relaxation of the sphincter. If this does not occur and the sphincter fails to relax the bolus swallowed is driven against the weak points in the posterior wall where frequent repetitions

of this abnormal occurrence may give rise to a mucosal herniation resulting ultimately in the formation of a pulsion diverticulum. As the bolus descends further, the mouth of the esophagus closes again.

The speed at which the material traverses the various portions of the esophagus depends to a considerable extent upon the characteristics of the muscle layers in them. In the upper third, where the muscle fibers are striated, it is rapid. From that level downward the progression becomes more and more leisurely as the bolus passes into the portion where there is nothing but smooth muscle. Although the esophagus of an individual swallowing a given mass of food behaves in an almost identical manner upon repetition of the act, two normal subjects may present striking differences in the evolution of the swallowing act and the length of time involved may vary considerably.

During deglutition there is usually a temporary delay at the narrow segment behind the aortic arch, the duration of which depends upon the physical properties of the material swallowed. This arrest is hardly perceptible for liquids but may be several seconds for semisolids or solid matter, depending upon whether the latter has been masticated thoroughly or not. In the latter case, solids may take as long as thirty seconds to pass this point. As the material descends below this point, it can be shown by fluoroscopy to outline the bronchial constriction. It is subject to the motions of the left atrium of the heart and then gradually accumulates at the level of the esophageal hiatus in the region referred to by roentgenologists as the 'phrenic ampulla'.

Two theories have been advanced to explain the mechanism by which material is propelled through the esophagus. In one the organ is considered to be a relatively inert tube through which material swallowed passes, largely because of its weight. But if the effect of gravity is thought to be all-important, it is hard to explain why liquid or very soft pastes may descend all the way to the diaphragmatic level following a single impulse from the pharynx, whereas the progression of semisolids or solids is often long delayed. In the case of the latter, the propulsive effect of the pharyngeal muscles is effective to the level of the aortic or bronchial constriction, but from this point down the peristaltic action of the esophageal musculature takes over. This activity is best seen fluoroscopically with the subject in the horizontal position, where these peristaltic movements become very obvious from the aortic narrowing all the way to the lowermost portion. In fact, the shadow of the ingested material often swings from side to side like the motion of a snake as it progresses downward. This motion is frequently relatively rapid, consuming no more than one or two seconds of time.

Esophageal Peristalsis

A careful analysis of the peristaltic activity of the esophagus suggests that it actually occurs in two principal waves. The *first* wave arises at the pharynx and passes all the way to the lower end. The *second* wave in contradistinction arises at the bronchial or aortic constriction and is propagated likewise to the lower end. The influence of this secondary wave is felt principally in the presence of a large bolus or one insufficiently masticated and mixed with saliva. It

may at times be difficult to differentiate these two waves, which may appear as one. During the passage of the first wave of propulsion the intraluminal pressure may rise as high as 10 mm. of mercury and the contraction may last as long as twenty seconds. The second wave or phase may reach a pressure of as much as 100 mm. of mercury. Its progress is usually at the rate of about 2 cm. per second. This wave, which is the result of smooth muscle peristalsis, is involuntary and is unaffected by functional disturbances of the striated or pharyngeal muscles as in bulbar poliomyelitis or in cerebral hemorrhages involving the swallowing center.

In persons over fifty years of age a *third* wave may be observed. This wave is often poorly defined. It tends to start at the level of the aortic arch and does not progress the entire length of the organ. Its action is short in duration and tends to be segmental or localized in extent.

The existence of these waves of contraction may explain the segmental spasticity of the esophagus sometimes observed, which when particularly pronounced, especially in older people, gives rise to the roentgen appearance often referred to as 'curling of the esophagus.'

The nature of the mechanism whereby the passage of food through the lower esophagus takes place has been the subject of much discussion. This has centered chiefly around an explanation of the fact that material is held back at a point about 2 or 3 cm. above the cardiac orifice of the stomach as if there were a sphincter at that point. Various theories have been advanced such as the influence of the phases of respiration, the constricting effect of the diaphragm at the esophageal hiatus, or the actual presence of an intrinsic sphincter muscle. None of these appears to be valid. There is no cardiac sphincter. In fact, the free regurgitation of material from the stomach into the lower esophagus can usually be demonstrated in any normal subject especially in the Trendelenburg position. The only reasonable explanation of the hesitation in the passage of material at this level is the sphincter-like tonicity of the smooth muscle of the esophageal wall aided perhaps by the few striated fibers often present.

The behavior of the esophagus at its lower end and of the cardia is of special interest. Although no true sphincter or external sphincter-like mechanism can be demonstrated the physiologic activity of the muscle layers of this portion serves to control to some extent the passage of material into the stomach. As the peristaltic wave reaches this area where the phenomenon referred to by the roentgenologists as the phrenic ampulla occurs, there is a peak rise in pressure with a plateau-like falling off to normal similar to that which prevails in the main portion of the esophagus as previously described. The wave at this level however proceeds much more slowly than above. It is unaffected by the presence of a hiatus hernia and significantly does not show any alteration of character in the presence of a paralysis of the left half of the diaphragm.

In the lowermost segment of the esophagus corresponding to the portion in and below the esophageal hiatus of the diaphragm the peristaltic activity is distinctly different. This is the portion referred to by some as the vestibule and by others as the cardiac ampulla. Here there is no forceful peristaltic wave but merely a gradual rise and fall of pressure of no more than 15 mm. of mercury, corresponding to the accumulation and subsequent passage of eso-

phageal contents This activity depends upon intrinsic reflex peristalsis and is characteristic of this segment only Although locally independent of the activity of the upper portions of the esophagus, the activity of this segment is integrated with it because, although normally in a state of tonicity, it relaxes as the main peristaltic wave reaches it from above

These observations based upon recent studies of the intraluminal pressures during deglutition help to explain the phenomena observed at fluoroscopy, particularly of the distensible segment just above the diaphragm termed the 'phrenic ampulla' This corresponds to the region in which the peristaltic waves are less forceful or absent While watching the passage of a bolus of barium meal, one cannot always distinguish the portion of the esophagus between this segment and the cardia

In addition to the peristaltic activity stimulated by the mere presence of food, the control of the lower esophagus and cardia may be influenced, as shown by Cannon many years ago, by the relative acidity (or pH) of the material swallowed and the gastric contents If the latter are neutral or alkaline, a rhythmic relaxation occurs which permits regurgitation from the stomach into the lower esophagus If the gastric contents are made normally acid, this regurgitation ceases after a short latent period This effect is not altered by bilateral supradiaphragmatic vagotomy or in animals by transection of the spinal cord in the thoracolumbar segment This phenomenon depends upon a locally acting reflex activity of the smooth muscle in this area and is further evidence of the presence of a more or less independent nerve muscle mechanism in the lower esophageal segment which serves as a physiological sphincter

Of further interest and importance to the clinician is the effect upon the function of the esophagus of responses to painful or unpleasant stimuli arising in other parts of the body, or of emotional disturbances Noxious stimuli such as immersion of a hand in ice water or painful pressure upon the scalp may in certain susceptible persons cause a delay of as long as three minutes in the passage of barium from the pharynx to the cardia The same effect may be observed as a result of emotional disturbances, worry, anger, etc The effect of emotional stimuli is observed classically as the *globus hystericus* or, more frequently and of more importance, as transitory exacerbations of dysphagia in the presence of constricting organic stenoses Thus a patient with a carcinoma of the esophagus who is still able to swallow liquid nourishment may experience a sudden total obstruction as a result of the receipt of unpleasant news, anger, or other emotional disturbances It cannot be said, however, that emotional stimuli even in achalasia, are sufficient to bring about any structural pathologic changes

Impulses of Extrinsic Origin

In addition to its peristaltic activity the wall of the esophagus is subject to movements produced by the transmitted pulsations of the heart and aorta, the phases of respiration and even of phonation The effect of the respiratory changes in intrathoracic pressure is of special interest Throughout the inspiratory phase the lumen of the esophagus remains patent During expiration the

lumen contracts. With the mouth of the esophagus closed, as in the resting state, these effects are least noticed in the neck and most pronounced in the mid-portion of the organ. Contrariwise, the motions of the esophagus induced by phonation are felt chiefly in the upper portion and are independent of the respiratory variations of intrathoracic pressure.

The Nervous Control of Deglutition

The swallowing center lies in the floor of the fourth ventricle of the brain above the respiratory center with which it has no connection. It is composed of several portions corresponding to the functions of the various regions involved in the act of swallowing, namely the pharynx, the several segments of the esophagus and the cardia. The function of this bulbar center is reflex. Rethi has, however, described a cortical center which by means of connecting fibers through the inferior portion of the internal capsule provides the mechanism for voluntarily activating the reflex swallowing mechanism.

It is thought that the primary and secondary waves of peristalsis in the esophagus are mediated through independent parts of the swallowing center. Peripheral stimuli which may set the reflex in motion arise in the base of the tongue, the soft palate, the posterior surface of the pharynx, the superior margins of the larynx, and in the upper esophagus. Excitation of the sensory fibers of the superior laryngeal nerve has been observed to initiate reflex swallowing. The principal zone of excitation of the reflex is the lower pharynx and upper larynx. If this region is rendered insensitive, the swallowing reflex is inhibited. Another stimulus which tends to excite reflex deglutition is distention of the esophagus, but it has been found that this form of stimulation is most effective in the upper third. In the lower portion it tends to produce merely local peristaltic contractions.

Little is known about other intricate ramifications of the reflex nerve pathways. That there is a communication between the esophageal and bronchial reflexes is, however, suggested by the clinical observation often made in patients with a hiatus hernia of the sliding variety in whom the temporary lodgment of food in the lower esophagus induces reflex coughing and choking even in the absence of aspiration of regurgitated material.

The motor impulses of the mechanism pass by way of the trigeminal, the glossopharyngeal, the hypoglossal and most important of all the vagus nerves.

As is well known, the effect of excitation of the vagus nerves on the alimentary canal as a whole is to provoke hypertonicity and peristaltic activity of the musculature with relaxation of the sphincters. This phenomenon as it affects the esophagus has been studied in man by observing the contrasting effects of drugs like atropine or Banthine and pilocarpine or more recently the cholinergic drugs of which acetyl beta methylcholine chloride (Mechoyl) is an example. The former have an antagonistic effect upon the activity of the nerve and the latter have a stimulating or vagotonic action. When atropine or Banthine is administered, the length of time required for the passage of swallowed material through the esophagus is prolonged by at least twelve to fifteen seconds because it eradicates the normal peristaltic wave. As might be anticipated after such an

tinal in origin and is usually caused by periesophagitis from inflammatory lesions of the esophagus, infected or penetrating carcinoma, or perforation of the esophageal wall with complicating mediastinitis

Esophagitis and particularly ulceration of the esophagus often cause intense substernal pain like that of peptic ulcer occurring elsewhere but referred to a different location

The deglutition of unusually hot or cold materials is readily perceived. Heat may be felt all the way down. Cold, however, is usually felt mostly at the lower end and is referred to the epigastric region in only a few seconds after the material is swallowed

A peculiar and little understood sensation which emanates from the esophagus is the burning discomfort experienced in the upper esophagus and pharynx, commonly spoken of as "heartburn" or pyrosis. This sensation arises from the esophagus, particularly in its lower portion. It is not necessarily actuated by the regurgitation of acid gastric juices, though this may be one stimulus which causes it. That this symptom is frequently due to distention of the lower esophagus has been shown by Jones and Chapman, who were able to reproduce it in normal subjects as well as in patients by inflating a balloon inserted at the proper level. In fact, it may be caused by stimuli of various sorts acting in the lower esophageal segment. Esophagospasm of any type or origin is a frequent cause of pyrosis. This sensation is a common occurrence in patients who have a hiatus hernia even though, as is usually the case, there is no associated esophagitis. Esophagitis is one of the several causes of this symptom, but the occurrence of pyrosis does not in itself indicate that an inflammation is present.

Other Esophageal Functions

The secretory activity of the esophageal glands depends upon the stimulation of the vagus nerves. Both types of glands contribute a thick mucous secretion which under strong stimulation becomes profuse and watery in character. The secreted mucus serves to protect the esophageal mucosa from abrasion by ingested food or other material swallowed. In obstructions of the lumen the amount of mucus poured out may be quite excessive. In fact, one of the classical symptoms of carcinoma of the esophagus is a constant welling up of mucoid secretion from the portion of the tube above the obstructing growth. During sleep the mucous glands appear to rest inactive.

There is no digestive function of the esophagus although during the passage of food through it the digestion of starch by salivary ptyalin (amylase) and maltase may begin. There is also a limited amount of churning of the food and some admixture with esophageal mucus.

Distant Reflexes of Esophageal Origin

Stimuli arising from the esophagus may under certain circumstances cause reflex responses in other organs and tissues. A notable example occurs sometimes on dilatation of a particularly tight stricture, when the patient may exert

ence lacrimation, salivation, hyperemia of the skin from vasodilatation, sometimes dermatographia, and elevation of the pulse rate with a transitory rise in the blood pressure. It has been suggested that this reaction, which is observed most often in young persons, may depend entirely upon vasodilatation brought about by the release of histamine or some similarly acting substance from the tissue trauma at the site of instrumentation.

CHAPTER 3

Roentgen Examination of the Normal Esophagus

Technical Considerations

In the normal adult the esophagus can rarely be visualized radiologically without the aid of a contrast medium because of the superposition of shadows cast by the interposed vertebral column, trachea, heart, aorta, and sternum, although occasionally a ribbon-like shadow which suggests its presence can be made out in the mediastinum.

In a few individuals who have developed the habit of swallowing air and in young infants in whom aerophagia is a normal occurrence, the esophagus can be clearly defined because of the contrasting shadow cast by the ingested air. This observation has been employed deliberately by causing the person to be examined to swallow an effervescent liquid containing a small amount of barium sulfate. This technique often makes it possible to obtain excellent views of the esophagus with clear definition of rugal folds and other surface irregularities on the interior. If a swallow of a thicker barium mixture is given soon after the ingestion of the effervescent mixture, a clear definition of the cardia can be observed as the heavy mixture pushes the gas through into the stomach.

The greatest dependence however, in the roentgen examination of the esophagus is upon the observation of radiopaque substances alone. These may be either solids, semisolid pastes, or liquids. The use of pills of barium sulfate mixed with a suitable substance to form a solid mass or of capsules containing Lipiodol is of historic interest chiefly. In practice, pastes or liquids are the most useful.

The use of a barium containing paste is the method of choice. A paste has the advantage that it imitates more exactly the form in which food which has been chewed and mixed with saliva is swallowed. A useful substance of this type can be made by combining a cooked cereal or a mixture of flour and water with

barium sulfate in the form either of a powder or of a cream made by diluting slightly with water

For a liquid medium, barium sulfate rendered relatively palatable by incorporating it in a chocolate drink or as one of the flavored mixtures obtainable commercially is usually employed. A colloidal suspension of a thorium salt gives excellent definition, particularly of the mucosal folds, after the bulk of the ingested fluid has passed on, but the probable danger of radioactivity following the absorption of some of this material has led to the abandonment of this technique by the majority of roentgenologists.

During the examination the subject should be close to the fluoroscopic screen (or to the cassette when taking films) in order to avoid distortion of the image. Because the esophagus lies nearer the posterior aspect of the thorax than the anterior, it will be found that the best position for oblique views is the left posterior oblique and for all others the cassette should be at the back rather than in front. For fluoroscopy the frontal position suffices.

In order to dissociate the shadows cast by the esophagus from those made by other organs and the bony structures of the thorax, it is necessary to obtain various views by shifting the position of the patient in relation to the machine. The optimal position of the patient for the examination varies somewhat depending upon which segment of the organ is of special interest.

In the neck the only obtrusive shadow is that of the vertebral column. Thus a lateral view is usually most helpful in this area.

In the thoracic region it is often necessary to evade the shadows of the aorta and heart as well as that of the spine. A lateral view gives a clear visualization of the esophagus behind the heart but in the supra aortic region the shadows cast by the shoulders obscure the field. It is difficult to eliminate these completely.

In general, oblique views are probably the most helpful. With them the heart shadow is projected on one side and the vertebral column on the other, leaving a relatively clear view between them where the esophagus lies. Furthermore, the shoulders are thrown out of position to some extent. In order to accomplish this to the best advantage, the right anterior oblique and left posterior oblique positions are used. Actually by testing various modifications of position under the fluoroscope the optimum placement can be determined for the examination of a given patient. Films are then made in that projection. The frontal plane of the patient should be approximately perpendicular to the direction of the rays. By combining a lateral exposure of the neck with the oblique view of the trunk an excellent view of the entire organ can be obtained on one film.

In the examination of the *lower esophagus and cardia* the frontal position (postero anterior) works well, but the oblique position is better because of the slight obliquity of the esophagus in this area produced by its swing to the left and forward as it approaches the cardia. In this area the shadow cast by the left lobe of the liver comes into view and must be differentiated. The air bubble in the stomach often provides an excellent contrasting shadow for the study of the cardia.

Sometimes, in order to gain a better impression of an abnormality in the

esophagus, it is necessary to slow down the passage of the radiopaque material through it. This can be accomplished in part by carrying out the examination with the patient in the recumbent position or even in a slight Trendelenburg position. In this position the organ tends to distend somewhat, the mucosal folds are likely to be better outlined, and diverticula, especially of the mid thoracic segment, are often better filled. When position alone is not sufficient, it is helpful to have the patient swallow a thick barium paste instead of the liquid mixture. The slower progression of this substance opacifies a longer given segment at one time and makes possible a superior delineation of surface irregularities of the mucosal lining.

Both fluoroscopic examination and the taking of films are essential in every instance. The former demonstrates the manner in which the esophagus fills and empties, the rate of passage of material, and the characteristics of peristaltic action. It serves also to indicate what views of abnormal areas are best obtained. The latter makes it possible to provide permanent views of what would otherwise be only transitory impressions and often brings to light certain details which were not observed at fluoroscopy. The motions of the heart and the occasional occurrence of rapid peristaltic waves in the esophagus itself make rapid exposures of the order of one-tenth of one second essential, with the subject not breathing and preferably at full inspiration. Serial views are particularly helpful.

Tomography has a limited value in the roentgen investigation of the esophagus, principally to aid in visualizing the relations of the organ to an adjacent mass in the mediastinum. It should be recalled, however, that this technique exposes the patient to an unusually large dosage of irradiation, the amount depending upon the number of films obtained. Its use, therefore, cannot be recommended except in unusual circumstances.

Roentgen Appearance of the Normal Esophagus

Because of the anatomical differences between them, it is necessary to give separate consideration to the roentgen appearance of the esophagus of the adult and that of infants.

The Adult Esophagus

The course of the esophagus is not exactly straight. In the frontal projection it describes a shadow with a double curve. The upper curve has its convexity towards the right beginning at the level of the seventh cervical vertebra, with its maximum opposite the third thoracic vertebra. After winding to the right of the aortic arch at about the level of the seventh thoracic vertebra it continues as an inverse curve with its convexity to the left as far as the cardia. In the sagittal plane the esophagus follows approximately the configuration of the spinal column but with a somewhat more obvious curve. Thus, although at its origin in the neck it lies close to the vertebral bodies, it ends at the cardia with a wide space between it and the spine which is occupied by the aorta and the left crus of the diaphragm (Fig. 33).

During inspiration the diaphragm, instead of sliding over the esophagus as

it descends, actually pulls the esophagus down by means of its normal attachments and tends thereby to straighten these curves

The *mouth of the esophagus* should be considered as a part of the study of the pharyngo esophageal segment. The radiological study of this segment is difficult in practice because of the rapid passage through it of the opaque bolus, which usually consumes $1/10$ to $5/10$ of a second to move from the base of the tongue to a point behind the clavicles. A special technique may be required involving the use of a somewhat thick paste of barium in order to slow the transit. It helps also to have the subject's head tilted slightly forward. Finally, it is necessary to have equipment which permits rapid exposures for the taking of the so called "spot films" of local areas. Likewise, it is indispensable to take

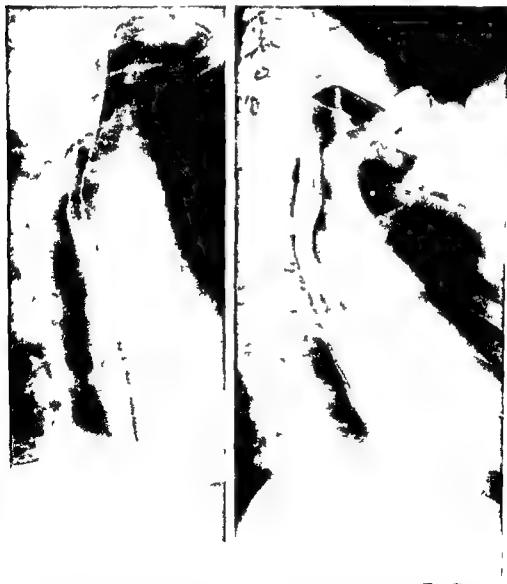


FIGURE 33 Normal esophagus Upright position right anterior oblique exposure



FIGURE 34 Mouth of esophagus (frontal view) Note the tennis racket shape of the shadow

films in the anteroposterior, lateral, and three-quarters oblique projections, with the segment filled at the actual moment of the passage of the barium mixture and immediately thereafter in order to study the mucosal folds as outlined by the small amounts of barium which lag behind. In the frontal position the subject's chin must be held midway between flexion and extension in order to avoid the interposition of the shadow cast by the mental symphysis or by the occiput upon that of the superior portion of the pharynx.

The subject should take a mouthful of opaque medium and should swallow it when told.

In the frontal (or postero-anterior) projection during the passage of a large swallow of barium, the shadow of the hypopharynx assumes a racket shape (Fig. 34). The image is rather dense and the details of the folds on the anterior surface are difficult to distinguish. Only the contours can be followed clearly. The body of the racket is formed by the fanning out of the opaque material over the base of the tongue and the posterior wall of the pharynx. The lateral contours are limited by the pyriform sinuses. The center of the shadow is uniformly less opaque because of the presence there of the epiglottis which arrests the barium to some extent. The handle of the racket represents the cervical segment of the esophagus just below its mouth, which lies therefore at the junction of these two shadows. However, no constriction can be observed and the caliber of the organ when completely distended shows a gradual diminution from the pharynx down into the esophagus. On the contrary, when it is not completely open, the mouth of the esophagus shows as a symmetrical hourglass constriction measuring approximately 1 cm. in diameter and lying in the average case opposite the fifth cervical vertebra.

Immediately after the passage of the opaque bolus, the folds of the anterior

surface of the pharynx become apparent as outlined by traces of barium which are left behind. The two superimposed transverse glosso-epiglottic folds and the epiglottis above arrest some of the opaque substance at the base of the tongue, causing a shadow which projects below that cast by the inferior maxilla. These structures form between them two small pockets shaped like pigeons nests, one on each side of the midline, which are called the *valleculae* or glosso-epiglottic fossae (Fig. 35). They are always seen and contain a portion of barium approximately 1 mm. in thickness giving an absolutely symmetrical image. At 1.5 to 2 cm. below the *valleculae* is another barium filled furrow. This extends horizontally and is slightly concave inferiorly. At times it is represented by two concave arches, one right and one left. This groove represents the superior orifice of the closed glottis. The double arch appearance of the shadow cast by this structure in some instances is caused by the escape of a little barium into the superior orifice of the glottis, which looks on the film like a little spur in the center dividing the arch in two (Fig. 35). At each end of this arch is a deep straight fold which fills with barium. These are the *pyriform sinuses*. They are joined on each side to the lateral extremities of the two *valleculae* by a thin streak of barium delineating the lateral walls of the pharynx (Fig. 35).

At the center of this pattern just described, which outlines the lateral wall of the pharynx is observed another shadow which leaves the *valleculae* and whose lateral walls have the appearance of a funnel. This represents the *hypopharynx*. The lower extremity of this funnel lies in the midline about 1 cm. below the *pyriform sinuses*. This corresponds to the mouth of the esophagus. It shows as a narrow point approximately 1 cm. in length below which the air filled lumen can be seen giving the appearance once again of an hourglass.



FIGURE 35 : Stasis in the *valleculae* and *pyriform sinuses*. This case of pathological stasis permits a study of the morphology of the region. (Note predominance on left side.)



FIGURE 36 Mouth of the esophagus in profile. On the anterior surface are seen from above down the outlines of the valleculae, the anterior portions of the pyriform sinuses, and the cricopharyngeus muscle opposite the discs of the fifth and sixth vertebrae.

In the lateral view, with the esophagus full, the shadow assumes a tubular appearance largest in its upper portion, where the oropharynx gives it the appearance of a funnel which slopes forward towards the buccal cavity. In this projection the mouth of the esophagus does not appear as a constriction in the shadow. The posterior wall is smooth and separated from the anterior surface of the vertebral column by the retropharyngeal space, which is several millimeters in width. The shadow cast by the anterior wall in this region is likewise relatively smooth, with merely two projections that are sometimes not very prominent. The upper of these lies at the base of the tongue and corresponds to the superior orifice of the glottis. It often assumes the shape of a triangle with its peak forward and its base facing posteriorly. The lower projecting shadow is linear and often has the aspect of an anterior reduplication of the barium column. This represents the pyriform sinuses, which join the hypopharynx at the back of the mouth. It should be mentioned that these two shadows lie on the posterior surface of the larynx, which is easily seen in this view. This grouping of shadows rises appreciably during the closure of the glottis, often as high as the third cervical vertebra (Fig. 36).

In the partially filled state the posterior wall appears as a rectilinear shadow in the prevertebral plane. The anterior wall shows the same irregularities, though less clearly than in the distended condition. At the inferior portion of the sloping pharyngeal surface of the tongue a little above the level of the hyoid bone, in the true lateral position, the valleculae can be seen, one superimposed upon the other, as a small opaque triangle pointing downward (Fig. 37). The shadow cast by the pyriform sinuses can be seen crossing the superior margins of the laryngeal cartilages. The posterior wall of the larynx is outlined by a vertical streak of opaque medium upon which lies the folds of the pyriform

sinuses. The outline of the hypopharynx, whose surfaces have just been described, terminates like the end of a funnel at the mouth of the esophagus behind the cricoid cartilage at about the level of the fifth cervical vertebra. An *oblique view* of this region is sometimes obtained in order to give better visualization of the valleculae and the pyriform sinuses, the pairs of which in the true lateral projection are superimposed one upon the other.

In the lateral view just below the termination of the hypopharynx at the mouth of the esophagus an indentation in the anterior surface of the barium shadow, caused by the presence of the cricoid cartilage, can be seen (Fig. 38).

The *constrictions which occur at the level of the aortic arch and left main bronchus* appear usually as a single narrowing opposite the third, fourth, and fifth thoracic vertebrae (Fig. 33, B). Sometimes depressions in the shadow corresponding to each of these structures can be made out. Often a thick paste of barium will pass by the aorta without hesitation, only to be held up momentarily at the level of the bronchus.

The segment of the esophagus lying behind the heart casts a shadow which is flattened anteroposteriorly by the left atrium which lies directly in front (Fig. 33, A). With this portion of the esophagus full of barium marked pulsations transmitted from the heart can be seen. These observations make it possible at times to study the volume of the left atrium and to some extent its function.

The *narrowing of the shadow which occurs as the barium reaches a point just above the diaphragm* corresponds to the location of the sphincter like lower segment of the esophagus. The roentgen appearance of this portion varies de-



FIGURE 37 Mouth of the esophagus (partial filling). The valleculae are represented by the spots visible below the mandible (A).



FIGURE 38 . Mouth of the esophagus
Note the depression on the anterior surface caused by the posterior margin of the cricoid cartilage

pending upon whether the musculature happens to be relaxed or contracted. In the relaxation which occurs as the barium mixture passes, the esophagus at this level dilates to equal the diameter of the thoracic segment, approximately 2 to 3 cm. The latter figure should be considered the upper limit of normal. The lumen appears tubular and smooth (Fig 39, A). Sometimes when the volume of barium passing through at the time is small the parallel longitudinal mucosal folds can be clearly seen in this area. When the musculature contracts between the passages of individual swallows of barium, this epicardial segment appears as a clear transverse band which separates the thoracic portion of the esophagus from the stomach (Fig 39, B). Two or three folds of mucous membrane containing barium may be recognized. They appear to be compressed together, but their presence often makes it possible to estimate the width and the length of the sphincter-like segment during this contraction phase (Fig 39, B). Its diameter is usually about 1 cm and its length about 3 cm. These dimensions correspond to those of the so-called diaphragmatic canal at the esophageal hiatus. Often this segment is represented only by a single, rather large fold of mucosa flanked by two small shadows representing a groove in the mucous membrane on each side. The upper part of this portion, shut off during contraction, may be sharply transverse when the esophagus is full of thick barium ready to pass through the area. But this shadow may be slender and tapering and may rise 1 cm or more above the diaphragm. From this point also the shadows of the rugal folds streak upward into the thoracic esophagus, where they are lost. The inferior portion of this segment during contraction is less easily demonstrated on roentgen films. It is located about 1 cm from the orifice of the cardia and dilates gradually from above downward to form the phrenogastric funnel. Often it is exactly at the cardia itself.

Because it is short, the abdominal segment of the esophagus may not

always be visualized, but in cases of gastroptosis it may attain a considerable length and be easily seen. This portion has been called by some roentgenologists the *cardial antrum*. It has a funnel shape like the end of a trumpet or the corolla of a petunia flower facing downward with its flaring opening at the cardia (Fig 40). The mucosal folds, after appearing to be squeezed together where the esophagus passes through the hiatus in the diaphragm, flare out in this funnel-shaped portion and provide contrasting shadows for comparison in abnormal conditions. In certain instances this region can be visualized to best advantage when the subject is placed in an accentuated Trendelenburg position while lying on his back. This is particularly possible if this segment of esophagus is open when it can be seen as a bubble of air in films taken in the erect posture. It can be visualized as well for purposes of special investigation by means of rubber balloons filled with opaque liquid.

Investigation of the *cardial orifice* can be easily accomplished by viewing its endogastric aspect using a double contrast technique with air and barium. The air pocket is usually most effective when the subject is erect, although views made in the supine position with or without the use of Trendelenburg tilting and in the ventral position with slight oblique rotation are also helpful. The cardia may often be seen as a gaping opening when the patient is not swallowing. Its inferior margin is grooved by the passage over it of the four esophageal mucosal folds which are continuous with the longitudinal folds of mucous membrane along the lesser curvature of the stomach (the gastric canal or "Mag-



FIGURE 39 The diaphragmatic portion of the esophagus A While open B during closure



FIGURE 40 Phrenogastric funnel (cardial antrum) at the lower end of the esophagus



enstrasse") The superior margin often is outlined by an arching fold of mucous membrane with its concavity downward. This has been referred to by certain roentgenologists as "the supracardial valve." Each end of this fold is lost in the lateral walls of the orifice (Fig. 41).

Thus, when viewed from below, the cardia looks like a star surmounted by a valve-like collar forming a semicircular arch which is more or less closed depending upon whether or not the gastric contents tend to impinge upon the lesser curvature of the stomach (Fig. 41, B). Its diameter varies from 0.5 cm. in repose to 2 or 3 cm. when fully distended by the passage of barium, thus never assuming a size greater than the diameter of the esophagus itself. Its relative proportions vary to some extent, depending upon position and the angle from which it is visualized.

The erect posture with the subject turned slightly in the right anterior oblique position is most favorable for the visualization of what has just been described. In this position the shadow of the cardia is projected against the gas-filled posterior portion of the fundus of the stomach and a good contrast is thereby obtained.

In the supine position the fundus is filled by the barium, but by choosing a certain degree of rotation in either the right or left anterior oblique positions, depending upon the circumstances, a view of the cardia in profile may be obtained (Fig. 42). If the esophagus is then filled by a swallow of barium, the *cardial incisura*, which corresponds to the angle of the esophagus with the adjacent portion of the fundus of the stomach, can be demonstrated. This angle on the exterior of the stomach denotes the location of the cardiac orifice within. In the position mentioned, this angle appears acute. Under certain pathological conditions it may appear to be widened. It is important to identify it in certain cases of hiatal hernia of the diaphragm.

If the esophagus is empty, the ends of the esophageal mucosal folds assume

■ serrated aspect around the gastric margin of the opening as outlined by the remains of the barium still adherent to them. This appearance, as mentioned above, is caused by the emergence of the esophageal rugal folds and, when the folds are abnormally thickened, may be pronounced. Usually one of these barium shadows is more obvious than the others, corresponding to the valvelike fold when open, thus allowing a little of the barium to remain in the funnel-shaped lower esophagus (Fig. 42)

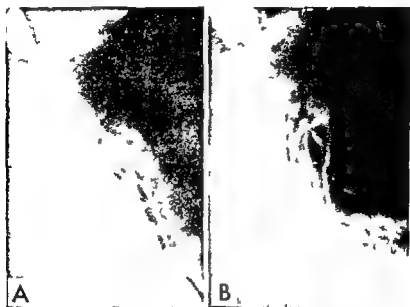


FIGURE 41 Valvular folds of the cardia frontal views (Jutras)



FIGURE 42 Profile view of the cardia



FIGURE 43 Cardial knob



FIGURE 44 Major fold of the esophagus Note pharyngo-esophageal diverticulum in the usual location

In the prone posture with slight rotation into the left posterior oblique position the visualization of the cardia is often inadequate because the mucus and gastric secretions which float on top of the barium accumulate in this region and prevent the obtaining of good mucosal relief.

In certain instances one can see a rounded gap in the barium shadow varying from 1 to 5 cm in diameter, which is often mistaken for a tumor. This image is circular with the starlike orifice of the cardia in its center. Although the reason for their assuming the configuration which gives rise to this image is not apparent, the structures of which it is comprised are as follows. The upper half of its circumference is usually circular and is marked by a thin border of mucous membrane. This represents the mucosal groove which surrounds the base of the supracardial valvelike fold previously described and separates it from the first fold of the pouch of stomach representing the upper part of the fundus. The inferior half of the circumference of this shadow is broken by the passage of the principal mucosal folds of the lesser curvature. This shadow is pushed slightly forward by the adjacent gastric wall and is best seen with a light coating



FIGURE 45. Major fold of the esophagus showing that it resists better than the others any constriction. The fold may actually increase its volume temporarily by swelling of the subjacent submucosa (Jutras)



FIGURE 46 View showing the continuity between the folds of the esophagus and the straight smooth folds of the lesser curvature of the stomach (the Magenstrasse) (Jutras)

of barium, thus giving the defect in the barium shadow which it is so important to recognize in order to prevent an occasional unnecessary exploratory operation with a preoperative diagnosis of neoplasm of the cardia. This appearance might be referred to as the *cardial button* or *knob* (Fig 43).

The *mucosal folds of the esophagus* in each of the three important levels (the mouth, the thoracic segment, and the epicardial area) can be clearly defined only by using small amounts of barium. The appearance of the folds at the mouth of the esophagus has already been described. It should be added that the *longitudinal mucosal folds* of the entire organ originate at that point, where they are squeezed closely together, but they spread out rapidly from that point down to assume their typical appearance in the subjacent portion. Normally there is no evidence of a *transverse fold*, although such shadows are seen in persons with the Plummer-Vinson syndrome.

The *longitudinal folds* of the thoracic portion cannot be visualized if the organ is distended. In fact, if its diameter exceeds 1.5 cm. the folds vanish and the mucosa appears perfectly smooth. When traces of barium remain adherent to the mucosal furrows between them, the folds can be observed before they

are caused by the activity of the peristaltic contractions. Under these conditions one usually sees one or two, sometimes three mucosal folds which descend the entire length of the esophagus to the epicardial segment (Fig 44). One fold is usually more pronounced than the others and is the last to disappear when peristalsis begins. This fold often appears to pass all the way through the lower segment to the cardiac orifice. It is known as the "major fold of the esophagus." It lies in the anterior wall of the organ and is continuous below with the mucosal fold on the lesser curvature of the stomach. Sometimes there are two parallel major folds instead of one.

In the segment just above the cardia the rugal folds are squeezed together at the point where it lies in the esophageal hiatus of the diaphragm, but from that point they flare outward concentrically in the funnel shaped termination of the organ. At this point the major fold assumes a slightly oblique direction because of the rotation of the stomach below to the right. At their passage through the cardia the folds of the anterior wall scarcely indent the supracardial portion (Fig 45). The major fold (or folds) which descends at this point on the right wall of the esophagus is here flanked by two other parallel folds all of which indent the otherwise smooth inferior margin of the cardia and extend all the way along the lesser curvature of the stomach to the pylorus to form the four folds of the Magenstrasse (Fig 46).

The Esophagus of the Infant

In children over two years of age the morphology of the esophagus from the roentgenologic standpoint does not differ from that of adults. In infants, however, particularly those still nursing the appearance is very different and quite characteristic.

Aerophagia is a normal occurrence in the nursing infant. Air is swallowed in great abundance during nursing and once swallowed it tends to regurgitate into the esophagus from the stomach because at this age the sphincter-like action of the lower esophageal segment is exceptionally weak (Fig 47, B).

The infant is examined without preliminary preparation. It is given a nursing bottle containing a normal ration of milk to which has been added a sufficient quantity of barium sulfate. Spot films are indispensable and the use of a grille is not necessary because of the small size of the subject. The child is examined both in the erect position and while lying down. In the latter position less air is swallowed.

The esophagus usually appears like a large pouch for its entire length and often as wide as the vertebral column. Its margins are smooth and straight although slight irregularities in contour are sometimes seen. It contains air to a greater or lesser extent (Fig 48). Usually the column of air is segmented into two or three large elongated bubbles, one superimposed upon the other and separated by opaque transverse bands of barium (Fig 48 5 6). Sometimes the esophagus is completely full of air and since its inferior portion is often dilated, takes on the appearance of a club. The contours of this shadow are outlined by a thin line of barium adherent to the mucous membrane. Its superior portion beneath the mouth of the esophagus is covered by a shadow of barium which looks like a little hood. Its lower aspect is covered just above the level of the



FIGURE 47 Esophagus of a nursing infant A Clublike appearance, B, normal aerophagia

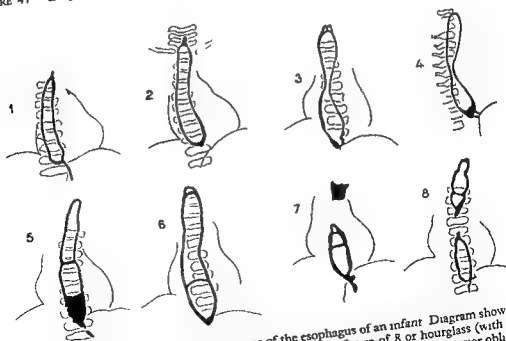


FIGURE 48 Roentgenologic appearances of the esophagus of an infant Diagram showing various shapes 1 like a club or leech 2 sausage shape 3 figure-of 8 or hourglass (with the cardia open) 4, hourglass shape with a bubble of air in the cardia (slight right anterior oblique position) 5 multiple air bubbles with indentations of the shadow in the epicardial segment 6, multiple air bubbles with base at the closed cardia 7, multilocular appearance with the middle portion pinched off 8 multilocular appearance (After M Leiong and P Aimé)

diaphragm by a residual of barium shaped like a cup (Fig 48, 3, 6) At other times the image looks like a sausage, long and cylindrical, uniform in caliber throughout and with a concavity towards the left (Fig 48, 2) Occasionally one may see two or three superimposed pockets of air outlined by barium and separated completely from each other by waves of peristaltic contraction (Figs 48, 8, 49, 3) Rarely a long extent of barium shaped like a ribbon or drumstick may be seen This may be due to extreme esophagospasm with failure to fill completely (Fig 50)

Finally, it should be noted that the imprint of the aorta upon the shadow cast by the esophagus is rarely seen in an infant For this reason it is sometimes difficult to estimate the diameter of the aortic arch

If one takes the trouble to study the lower esophageal segment by serial films, it is possible to observe the same behavior as in the adult with the exception that in the infant frequent regurgitation from the stomach is seen From the anatomical and roentgenological points of view this segment is normal, but from the functional viewpoint it is incompetent, especially in the supine position Thus one might think of the esophagus of an infant as a primordial gastric pouch

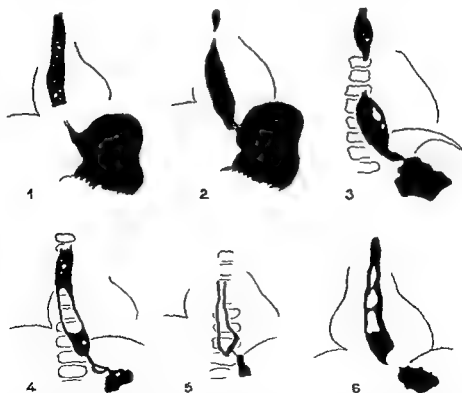


FIGURE 49 Roentgenologic appearances of the infant esophagus 1 Examination in the recumbent position folds of the half-open lower segment shown 2 examination in the recumbent position cardia slightly open 3 4 deglutition in the upright position cardia open 5 contraction of the portion above the cardia 6 cardia closed tight (After M Lelong and P Aumé)

Fluoroscopic Observation of the Swallowing Function

It is important to realize that the swallowing of solids differs from that of liquids and that there are differences of appearance between single swallows and repeated associated or successive swallows

Deglutition of Solids

If a solid object such as an opaque pill is given without a swallow of water to assist its passage, it will be seen that the effort of the pharynx to propel it is somewhat difficult. The pill at first moves back and forth and then suddenly passes from above down and into the mouth of the esophagus. The first swallowing motion pushes it a variable distance, sometimes as far as the manubrium of the sternum, sometimes to the level of the aortic arch, but rarely beyond. When the pill stops in the cervical segment, the subject always experiences a sensation of discomfort which causes him to make several more or less effectual swallowing motions until it reaches the aortic arch. The passage through the cervical esophagus is rapid when it results from a single effort of deglutition, but when several efforts are required, the speed is considerably diminished.

The progress of the pill is always arrested at the level of the aortic arch. If the subject continues to swallow his saliva, the delay is of short duration. If, on the other hand, he avoids further swallowing motions, the delay at this point may last as long as thirty minutes. Usually the resumption of its downward passage requires the action of a complete motion of swallowing effort which dislodges it in about three to four seconds.

The time required, however, for a pill to traverse the entire esophagus is exceedingly variable. When the subject makes repeated swallowing motions, it is no more than two to three minutes. If no further swallowing motions are made after the pill has entered the esophagus, it may be as long as thirty or forty minutes. In this instance the moments of progression are extremely short (a few seconds at the most), while the periods of arrest are as long as ten, twenty, or even thirty minutes. When the subject assists the swallowing of a pill with sips of water, complete passage rarely requires more than four minutes.

In other instances, especially if the subject is not young, a certain deeply felt sensation resembling the beginning of eructation is experienced. At this moment the pill will at times pass back up the esophagus several centimeters, but more often it resumes its downward course. Sometimes it will pass through the entire lower esophagus in a single sweep. Exceptionally small pills appear to be falling through an air-filled space, but with others the movement is slower. Sometimes the pill stops behind the heart for a variable period of time, where it may be seen to move synchronously with the heart beat. The arrest of a pill at the cardia is unusual.

The swallowing motions made while the pill is lodged temporarily in the esophagus produce varying results. Some produce no effect. Others push the object an almost imperceptible distance. What appears to be most important is the amount of saliva swallowed with these motions rather than the presence of the wave of contraction of itself.

When the pill lodges at any given point above the aortic arch, its presence

can be felt. This gives rise to an abundance of salivation and an urgent desire to swallow. Sometimes, however, this sensation may persist even after the pill has reached the stomach.

From the roentgenological viewpoint, however, the observation of the deglutition of solid material does not give sufficient information about the behavior of the wall of the esophagus. In order to accomplish this, pastes or liquids must be employed.

Deglutition of Pastes and Liquids

PASSAGE THROUGH THE HYPOPHARYNX AND THE MOUTH OF THE ESOPHAGUS. On the base of the tongue the shadow of a bolus of paste or liquid is large, round, and a little flattened, clearly defined and somewhat compressed towards the midline. Immediately below this level it flattens out symmetrically throughout and thereby becomes more transparent as it is compressed. The shadow then divides into two narrow concave lateral components which flow around the larynx to pass back and downward. Further on, the two currents approach each other again to meet in the midline where they are sometimes slightly separated by a thin elongated gap. At the mouth of the esophagus they always unite to form a single shadow. What happens is that, as the bolus falls into the valleculae, the epiglottis acts like a breakwater, forcing the current of material against the sides of the pharynx. There is little difference in this respect between the deglutition of liquids and that of solids.

With liquids, as we have just said, the fluid stream sliding over the base of the tongue is generally divided into two equal parts by the closed epiglottis and flows into the valleculae and the pyriform sinuses as far as the mouth of the esophagus where the two streams join. However, a slight amount of asymmetry of the sides cannot always be considered indicative of a pathological condition.

With pastes the division may be quite asymmetrical without having a pathological significance. Sometimes one side, sometimes the other, may predominate and in some subjects more material always passes down one side than the other. In the supine position asymmetry of the shadow is the rule. Although the pattern within the hypopharynx may appear to be distorted, above all at the beginning, once the act of swallowing is accomplished the field is entirely clear, completely evacuated as though swept clean.

Often after the principal portion of a bolus has already been swallowed a thin covering of barium can be seen sliding slowly over the convexity of the base of the tongue and becoming diverted into the lateral gutters. Once the latter are filled, an additional movement of deglutition starts up, sweeping the pharynx clear of all remains of the swallowed material. This becomes pathological probably only if it lasts more than ten to fifteen seconds.

This perfect evacuation of the recesses and gutters is probably the result of a complicated peristaltic activity which is still imperfectly understood. In any event it emphasizes the narrow interdependence between mechanical situations and reflex mechanisms. Thus the slightest disturbance, be it nothing but a simple hypesthesia of the mucous membrane, gives rise immediately to the principal sign of an abnormality in this region, namely stasis in the valleculae.

PASSAGE THROUGH THE CERVICAL AND THORACIC SEGMENTS It is convenient for the purpose of description to discuss separately single acts of swallowing as contrasted with repeated or successive swallows.

One or two single large swallows provide the best means of studying esophageal peristalsis. Three kinds of peristaltic waves can be distinguished, namely the primary, secondary, and tertiary waves (mentioned in Chapter 2). Only the first two are observed in normal subjects. These will be studied here.

The *primary peristaltic waves* follow deglutition as initiated in the pharynx. They begin as soon as the propulsive effort of the pharynx has forced the barium beyond the mouth of the esophagus. They are best seen in the horizontal or slight Trendelenburg position because the effect of gravity is thereby abolished and the progression of the bolus is correspondingly slowed down. Sometimes it is best to put the subject in an extreme head-down posture for this purpose. It should be pointed out that the peristalsis of this wave in the portion of esophagus proximal to the aortic arch is somewhat different from that below the arch. In the former, probably because the muscle layer is composed largely of striated fibers, the contractions are rapid and the passage of material through this segment is swift. Sometimes it is difficult to follow it with the eye. In fact, it often appears as though this portion empties itself into the lower esophagus with a single impulse. Sometimes a small quantity of barium is held up for an instant above the aortic narrowing, but it is soon ejected into the esophagus below. In the portion below the aortic arch, which contains smooth muscle fibers whose contractions are much slower, plenty of time is available to study the peristaltic activity. It will be seen that the swallowed barium assumes a fusiform shape and progresses relatively slowly towards the cardia. It is pushed along by a circumferential contraction of the esophagus which passes downwards at a regular pace. As it progresses, it opens up the lumen of the esophagus which cannot be detected until it appears (Fig. 50).

These primary waves may vary in intensity and in frequency. However, it is unusual to see more than two at one time throughout the entire length of the esophagus. On the average they require two to five seconds to reach the cardia. They may measure as much as 10 cm. in length and are separated by a distance of about 5 cm.

It should be mentioned that, especially in the horizontal position, the lower portion of the esophagus may remain filled with barium for as many as a dozen seconds without any peristaltic waves being seen. At the end of this quiet period, however, the contractions reappear and empty the esophagus of its contents.

After the passage of the bulk of the barium the esophageal mucosa may remain impregnated with small amounts of barium for a period of time, during which the longitudinal mucosal folds may be clearly seen. Sometimes a bubble of ingested air surrounded by barium may assume likewise a fusiform shape. But new peristaltic contractions arrive to sweep the esophagus clean.

The *secondary peristaltic waves* arise and remain solely within the lower third of the esophagus. They are characterized by the tendency to propagate both cephalad and caudad at one and the same time. They are not, however, sufficiently intense to be considered antiperistaltic or actually to border on regurgitations. They are observed only in the lying-down position, when they

may sometimes bring about a backflow of barium as high as the supra-aortic portion of the esophagus, but when this occurs, a mass contraction of the proximal third develops to push the opaque material back again into the lower portion.

These secondary waves arise when there is a somewhat prolonged hesitation of the bolus above the cardia. They are caused by the local excitation of the intramural centers of the esophageal wall produced by stagnation of the barium there. They are propagated along the wall by the activity of the plexuses of Auerbach and Meissner. They are very frequent in organic lesions of the esophagus which tend to cause obstruction.

At this point it is important to call attention to the *an p dila of the esophagus*. Although certain authors consider it to be pathological or at least a manifestation of senescence of the esophageal wall, this phenomenon is observed so frequently in apparently healthy people that it is more reasonable to consider it a part of the peristaltic activity of the normal esophagus. Very frequently in fact especially with the subject lying down a certain amount of barium becomes blocked between the lower or epicardial segment, which is delayed in opening, and a peristaltic wave above, which is sometimes rather forceful in the supine position. Under the effect of these two contrary pressures the esophageal walls become distended and the pocket thus created assumes a spherical shape with a diameter sometimes as large as 5 cm (Fig. 51). This phenomenon has several characteristics which should be noted. In the first place the dilatation is perfectly concentric in the long axis of the esophagus and therefore cannot be mistaken for a hiatus hernia or an epiphrenic esophageal diverticulum. Secondly, this bulging is transitory and at the end of several seconds it empties completely into the



FIGURE 50 Esophagus of the infant primary peristaltic wave giving rise to a fusiform appearance



FIGURE 51 Phrenic ampulla of the esophagus completely filled (adult) appearance suggesting the segments of an orange

stomach or disappears if the barium is pushed back up the esophagus by the action of a secondary peristaltic wave. Furthermore, it tends to reappear in the course of subsequent swallows. It is unusual, in fact, for this phenomenon to be observed as an isolated occurrence. Beneath this pocket lies the esophageal hiatus of the diaphragm and the cardia, both of which are absolutely normal although in certain instances a so called "enlargement of the cardia" can be seen. This is to be described further on.

Within this dilated area the mucosal folds of the esophagus can be seen distinctly and in this respect two types of shadow can be distinguished. The first is that where the ampulla is completely filled and perfectly spherical. In this instance the appearance is absolutely characteristic. The mucosal folds are, as it were, stretched out and enlarged at the point of maximal distention of the ampulla. Contrariwise, these folds are squeezed together above by the contractile wave and below by the action of the cardia. Thus they impart a fusiform appearance which looks like the sections of an orange after it has been peeled (Fig. 51).

If the degree of dilatation is less pronounced because of incomplete filling, it is effective only on one wall of the esophagus (usually the sloping wall). The ampulla then assumes a bilobar appearance across which one can distinguish the normal folds and furrows of the opposite wall extending as far as the cardia (Fig. 52).

In the course of its formation the ampulla of the esophagus takes on two particular attributes which are worthy of special description. The first is that at its inferior portion it may actually spread out so that it comes to rest upon the superior surface of the diaphragm. This produces at the base of the ampulla, surrounding the superior margin of the esophageal hiatus, the appearance of a

circular gutter filled with barium, which looks like the ring around the planet Saturn (Fig 53, *B*). The second is that at its superior portion there is a sort of circular mucosal fold which corresponds to an exaggeration of the physiological constriction of Von Hacker. This is due to a forceful infolding of the mucosal layer only, which becomes pinched between the ampulla below and a certain quantity of barium which is being forcibly pushed along by a peristaltic contraction above (Fig 53, *C, D*). This appearance has been described by Ingelfinger and by Schatzki and called a "contraction ring". It has no pathological significance. Both of these appearances are obviously transitory and disappear as the ampulla empties.

Certain authors have tried to ascribe to the esophageal ampulla the role of a pregastric pouch whose function it is to regulate and control the passage of food into the stomach. This function, however, must be of secondary importance as it exists primarily in the recumbent position, whereas eating is customarily carried out with the body erect. However that may be, it is in this portion of the esophagus that the reflexes which initiate the opening of the cardia arise.

Repeated Successive Swallows or Gulping. During the process of gulping or making repeated swallowing motions the esophagus takes on the appearance of a ribbon traversing the entire length of the posterior mediastinum and uniting the mouth of the esophagus with the cardia without loss of continuity. In the recumbent posture, however, distinctly demarcated primary waves of contraction can be seen undulating the esophagus but without breaking up the lumen into segments. In the erect posture the esophagus has a truly tubular appearance. The transit is very rapid and the walls of the organ do not appear to be affected by any peristaltic activity. A bubble of air may lie on top of the opaque column during these deglutitions.



FIGURE 52 Phrenic ampulla of the esophagus partially filled. The mucosal folds of the opposite wall are shown traversing the distended portion.

Stimulation of the gastric mucosa in the normal state tends to provoke the closure of the sphincter-like segment rather than to favor its relaxation. Another reason for the maintenance of this tonicity is the effect of ingested air which tends to inflate the fundus of the stomach, the medial portion of which in turn presses against the cardia causing a valve-like closure. Still another factor is that in the erect position the gastric contents tend to gravitate to the lower part of the stomach and in addition are forced upon the pylorus rather than the cardia by normal peristaltic activity.

It should be pointed out, however, that the actual orifice of the cardia is now and then wide open, but as a rule nothing from the stomach can pass beyond the level of the hiatus. In fact, by means of special studies one can demonstrate that the cardia is usually open and that by inflating the stomach this orifice can be dilated to look like an inverted funnel or horn of plenty. The prevention of regurgitation to higher levels, therefore, is actually a function of the sphincteric tonicity of the lower segment or "epicardia" of the esophagus.

Summary: Radiological observation of the esophageal phase of deglutition demonstrates the following sequences:

- 1 The alimentary bolus is propelled by the musculature of the oropharynx towards the mouth of the esophagus, into which its passage is accelerated because of the emptiness of the hypopharynx. As soon as the bolus clears the mouth of the esophagus, the latter closes as if hermetically sealed and the *primary wave of peristalsis* begins its progress towards the cardia.

- 2 In the thoracic esophagus the bolus continues to be acted upon by the primary peristaltic waves initiated by the contraction of the pharynx. These are forceful and symmetrical circumferentially as far as the segment above the aortic arch. Then, on leaving the region of the arch, the waves become slower and progress through the lower two-thirds. Finally the secondary waves of contraction can be seen working upon the bolus for a period of several seconds. These are initiated in the lower third and are provoked by distention of the esophagus.

In the lowermost part of the esophagus a so-called 'ampulla' can often be seen, especially with the subject in the recumbent posture. This ampulla acts somewhat like a reservoir to regulate the distribution of the swallowed material into the stomach. This function appears to be of little consequence in the normal erect posture where the effect of gravity is more marked. It is certain, however, that the local reflexes which cause opening of the lowermost segment arise in this zone.

- 3 After a more or less prolonged arrest in the lower segment (epicardia), the alimentary bolus overcomes the sphincteric mechanism in which the diaphragm, the diaphragmatic segment of esophagus, and the so-called subcardial valve or fold of gastric mucosa play a part in the closure. The muscle fibers of the fibromuscular fascial layer inserted in the diaphragm and the pressure of the bolus itself contribute to the opening of the segment.

Normally the sphincteric mechanism maintains an effective closure, but it is sometimes possible to visualize the phrenogastric funnel or actual cardiac opening, which is usually gaping. Inspiration and certain reflexes of gastric origin act upon the closure. Stimulation of the esophageal mucosa above the

lower segment (epicardia) and certain reflexes like those initiated by swallowing contribute to its opening.

4 Finally in certain subjects, notably those of advanced age a set of limited segmentalized tertiary peristaltic waves whose significance is not yet clearly understood, may occur. They are probably originated by the intramural autonomic plexuses. These waves are observed in the so-called 'curling' of the esophagus (Chapter 8, Fig. 153).

Deglutition of Liquids in Infants

Fluoroscopic examination of the esophagus of an infant while nursing reveals that a large amount of air is ingested during the process. With solids or semisolids, the aerophagia is less pronounced. The position occupied by the infant during the examination makes little difference. The appearance is essentially the same whether he is in the horizontal position or is held erect. During normal deglutition the opaque liquid progresses by the effect of esophageal peristalsis. The weight of the material alone often appears to be insufficient to bring about a normal transit.

In the nursing the peristaltic waves appear energetic. After eighteen months to two years the behavior of the esophagus during deglutition takes on the aspect of that of an adult. The disappearance of the infantile appearance and functional behavior coincides in part with the change in the method of alimentation with the abolishment of sucking and in part with the loss of the infant's tendency to regurgitate.

The esophagus must be empty In patients with mega-esophagus or with diverticula, appropriate measures by lavage or otherwise should be undertaken to evacuate the contents in order to avoid the sudden aspiration of esophageal contents into the respiratory tract in the event of sudden regurgitation For the same reason, the stomach should be made empty by withholding food and avoiding liquids during several hours before the examination

A preliminary sedative such as a barbiturate or morphine if preferred should be administered If salivation appears to be excessive, *atropine* should be given

There is no unanimity of opinion regarding the use of *anesthesia* during the procedure Some endoscopists still insist upon general anesthesia, but it is bothersome and not very helpful The experienced operator does well without it whereas another with less experience will encounter difficulties even with its aid In adults, if anything is required, local anesthesia is preferable

Reflex nausea is usually experienced by the patient as the instrument passes the isthmus of the cricoid cartilage, but ordinarily these reflexes subside once the tube has been introduced into the esophagus It is better, therefore, from the patient's point of view to precede the examination with topical anesthesia The procedure can then be done with greater accuracy and with less disturbance This anesthesia must, however, be administered with care Cocaine hydrochloride (1 to 20 or 1 to 10 dilution) with Adrenalin added is employed to reduce the sensitivity of the mucosa of the hypopharynx The esophageal mucosa, one should remember, is insensitive Alcoholics may require a dose of morphine

In addition to the pharyngeal mucosa, it is necessary to anesthetize the orifice of the larynx and, above all, the aryepiglottic folds because the contact of the tube with these structures provokes reflex coughing The interior of the larynx need not be anesthetized Application of the cocaine to the two pyriform sinuses is superfluous because, by anesthetization of the pharynx, these parts become insensitive as well

With young infants it is possible to carry out an esophagoscopy without the use of an anesthetic if the help of trained assistants is available However, even though one can overcome the defense reflexes of the sick child by these means it is impossible to suppress spasm and reflex vomiting During spastic contraction of the esophagus, the lumen closes completely It is only during short inspiratory movements that the lumen opens enough to make it possible to see well enough to manipulate the instrument Under these circumstances the possibility of injury to the wall of the pharynx or esophagus is much greater than when the child is asleep under an anesthetic which abolishes all struggling and harmful reflexes For these reasons unless the examiner is exceptionally well experienced and skillful, it is wise to examine all infants under general anesthesia The anesthesia should be pushed to the point of disappearance of the reflexes induced by the introduction of the tube If the intervention lasts a long time and if the anesthesia begins to disappear, the simplest method of prolonging it is the employment of a tampon of cotton wadding or gauze soaked with ether or chloroform inserted over the mouth around the tube

With older more cooperative children an esophagoscopy can often be carried out under local anesthesia

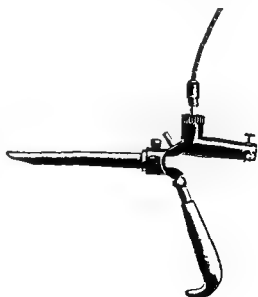
Instrumentarium

The technique of esophagoscopy depends somewhat upon the nature of the instrument employed. At present the instruments of Chevalier Jackson and of Haslinger are the most convenient, although for direct inspection of the mouth of the esophagus it is often best to employ a laryngoscope or the small esophagoscope intended for children as devised by Roberts.

The Haslinger esophagoscope is actually the instrument of choice. Because of the characteristics of its tip, shaped like a snowshoe, it is possible with it to avoid accidents especially during the passage through the mouth of the esophagus. It also provides good visibility. It has, moreover, undergone several modifications notably the elimination of the elongating tube which diminishes the effectual diameter of the lumen. By making the essential preliminary roentgenological examination the endoscopist learns in advance the location of the lesion and may then select a Haslinger tube of appropriate length. An armamentarium of tubes of various lengths and diameters, all adaptable to the electrosopic handle, should be available. The lengths of these tubes vary from 20 to 45 cm.

With the electroscope of Haslinger, in contradistinction to all other types of apparatus, access through the lumen is unimpeded and the operator is not hindered by reflections from its inner surfaces (Fig. 54). This instrument, which provides proximal lighting, is usually employed either with a Bruening tube which has a flaring tip and may be used for tracheoscopy or bronchoscopy as well, or with tubes the ends of which have rounded edges made especially for esophagoscopy. These edges are smooth and slide easily so that even in the most difficult cases one can avoid injury to the mucosa of the posterior wall during the passage through the constriction at the cricoid cartilage (Fig. 55). With ordinary tubes the edges are too often relatively sharp. Furthermore, the possibility of introducing these tubes with rounded edges almost without any danger

FIGURE 54 The Haslinger panelectroscope



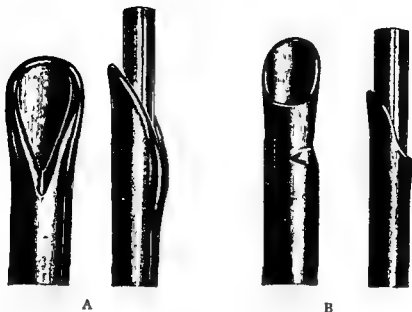


FIGURE 55 A The Haslinger tube showing the snowshoe shaped tip with and without the elongation tube (for adults) B, The Bruening tube with and without the elongation tube (for children)



FIGURE 56 : Esophagoscopic tube with snowshoe shaped tip devised by Haslinger with its elongation tube

of injury permits the use of tubes having a larger diameter. Also, one can employ longer tubes, measuring for example 50 cm, which make it possible to reach the cardia without having recourse to the intraluminal elongating tube which is indispensable with either the Bruening or the original Haslinger instrument whose outer cylinder measures only 25 cm in length (Fig 56)

The advantage of instruments having an elongating inner tube is that the operator works, whether during inspection or instrumentation, at the optimum distance because the tube can be introduced just as far as necessary in any particular case. On the other hand, with a long tube of fixed length when dealing with pathological conditions or foreign bodies lying in the esophagus, it is necessary to work from too great a distance. A constant objection to the use of intraluminal elongating tubes is the impairment of the field of vision which results when they are in place.

Relative to the choice of tubes to suit the age of the patient the following recommendations may be made. For children one to six years of age a tube 8 mm in diameter and 20 cm in length is used. For children older than six years and for adults a tube with a diameter of 10 mm and length of 25 cm is

employed. In each instance, of course, an elongating inner tube is necessary to reach the lower end of the esophagus. With patients who present unusually favorable anatomical conditions for endoscopic examination, such as lack of upper teeth and a flexible neck, in whom an easy introduction of the instrument can be anticipated, a tube 12 mm in diameter and 25 cm in length may be employed. Sometimes a 14 mm tube can be used.

The exterior tube 20 to 25 cm in length, which matches the distance from the upper dental arch to the aortic narrowing, can be used to examine the esophagus down to that point. If the lower esophagus is to be examined, the elongating inner tube is then introduced through the lumen of the first.

Techniques of Esophagoscopy

The Haslinger Technique

The Haslinger technique is adaptable to the use of either the sitting or the recumbent position. The advantages of the latter, however, are more obvious with esophagoscopy than with laryngoscopy. In the sitting position the accumulation of secretions is a source of great annoyance. Orientation and visibility are greatly impeded by the secretions and constant application of the suction apparatus is required. It is not only the secretions in the pharynx which complicate the examination, for the saliva from the mouth keeps running into the larynx and trachea to provoke the cough reflex. Furthermore, certain pathological conditions, as for example mega esophagus which is often attended by excessive accumulations of retained food and mucus in the dilated esophagus, provide insurmountable obstacles to the use of the sitting posture. A lavage of the esophagus which is often needed in such patients to make the examination possible, can be carried out only in the recumbent position (Fig. 57).

The optimal position for the head is the same as for direct laryngoscopy. This serves both for the introduction of the tube and the passage through the cricoid isthmus. The head is supported on a specially devised headrest at a level slightly above that of the horizontal plane of the table (Figs. 58 and 59). It is held by an assistant in maximum extension at the articulation of the occiput with the atlas (Fig. 60). In this position it is possible to carry out the examination as far as the aortic narrowing.

To see the inferior segment of the esophagus which describes a slight ventral curve beyond the aortic arch, it is necessary to lower the head of the patient progressively to a level below the plane of the table in order to follow the direction of the esophagus with the tube. For this manipulation the head may be held by an assistant or it may rest upon the headrest. If held by an assistant, his right hand supports the occiput while his left hand is used to elevate the angle of the mandible (Fig. 60), thus producing the hyperextension which is so important.

The entire examination, however, is more conveniently performed with the aid of the headrest. An assistant to manage the head is then not needed, relaxation of the muscles of the neck is more complete, and every position which may be desired can be maintained with greater precision than by the hand of an assistant. The headrest employed is constructed in such a manner that all the

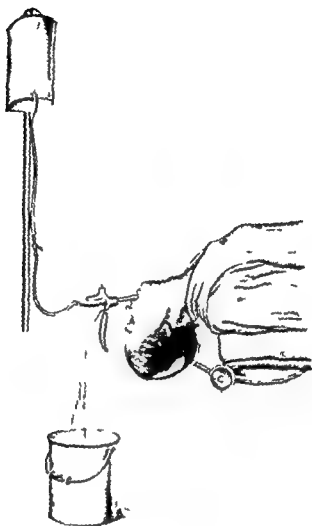


FIGURE 57 Lavage of the esophagus

movements of the head, whether lateral or anterior and posterior, can be executed easily (Fig 58)

Ordinarily the Haslinger esophagoscope is introduced in the midline. Introduction through the side of the mouth is useful in the edentulous patient or when there are unusually prominent central incisors which hinder the manipulations. The tube should be introduced in a systematic fashion and, if the following four steps are observed, success is assured

- 1 First, while the operator slides the tube along the base of the tongue as far as the epiglottis, an assistant pulls upon the tip of the tongue with a pledget of gauze (Fig 61)

- 2 Once the epiglottis has been recognized, with the instrument still in the midline, the tube is pushed down about 2 cm beneath the epiglottis (Fig 62)

- 3 The operator then directs the assistant to relinquish his hold on the tongue and, by adjusting the headrest, to move the head into the position of extension mentioned above. At the same time the operator lowers the handle of the electroscope and the tube then pushes the epiglottis and base of the tongue forward. The two arytenoid cartilages now enter the field of vision (Fig 63)

FIGURE 58 The Haslinger headrest. This apparatus is fastened to the table. The head can be raised or lowered by means of a ratchet and turned from side to side on a swivel mechanism. Many modifications are available.



FIGURE 59 Esophagoscopy in the recumbent position using the Haslinger headrest.

FIGURE 60 Esophagoscopy in the recumbent position using the Haslinger headrest. During the passage through the cricoid narrowing the head is placed by the assistant in maximal extension.



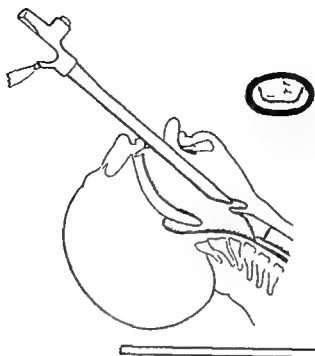


FIGURE 61 First step Visualization of the epiglottis (Oval insert endoscopic view)

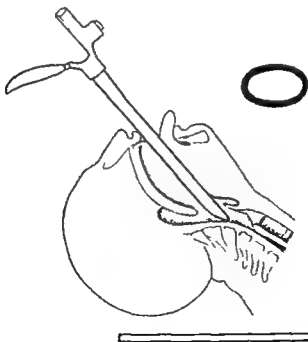


FIGURE 62 Second step The tube about 2 cm below the epiglottis in contact with the posterior wall of the pharynx (Oval insert endoscopic view)

These three steps therefore are comparable in every detail to those of direct laryngoscopy, but the *fourth is different*. In laryngoscopy and bronchoscopy, after having directed the tube towards the posterior wall of the larynx one lowers the handle of the endoscope still more and thereby presses the distal

extremity of the tube more forcibly against the epiglottis and base of the tongue. On the contrary, in esophagoscopy after the third step one executes an inverse movement with the distal end of the tube, namely towards the posterior wall of the pharynx.

4 Between the posterior wall of the larynx and the posterior wall of the pharynx lies a horizontal groove into which the end of the tube is made to pass after viewing the last landmark on the back of the larynx namely the two arytenoid cartilages. The tube then enters the first constriction which is the cricopharyngeal pinchcock or mouth of the esophagus (Fig. 64). From the clinical point of view this region is the most important of the whole esophagus because the vast majority of foreign bodies stop at this narrow portion.

The passage of the cricoid isthmus is the most difficult and dangerous part of the whole esophagoscopic examination. Most of the accidents which occur in this region are caused by wounding or actually perforating the posterior wall by the tube. These perforating lesions are followed by inflammation (often purulent) of the periesophageal connective tissue layer, and frequently this inflammation is followed by mediastinitis. Therefore to avoid this danger in the course of the passage of the cricoid isthmus, it is necessary to know the technique of introduction of the tube in every detail and above all the precautions necessary to avoid this grave lesion of the esophagus.

After the posterior wall of the larynx has been visualized the tube is slipped cautiously through the horizontal slit situated between the larynx and the posterior wall of the pharynx. Frequently it can be seen that this groove separates the field of vision into two parts one anterior, somewhat smaller and lighter, and the other posterior and darker (Fig. 64 a). The anterior portion

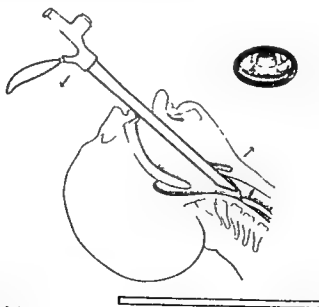


FIGURE 63 Third step. By means of pressure maintained against the tongue and the epiglottis the posterior wall of the larynx with the two arytenoids is brought into the field of vision. (Oval insert endoscopic view.)

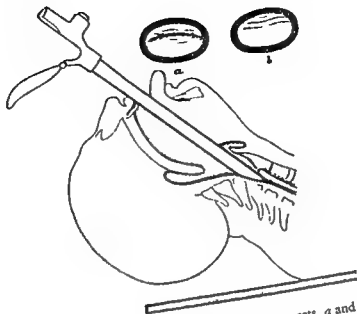


FIGURE 64 The passage of the cricoid narrowing (Oval inserts *a* and *b* variant endoscopic views)

corresponds to the dorsal part of the larynx, the posterior to the posterior wall of the pharynx. Sometimes, however, the above described groove is invisible and the entire field of vision is occupied by the posterior wall of the pharynx (Fig 64, *b*). Then, in pushing further forward the tube penetrates the groove like a wedge and displaces the mobile arytenoid cartilages forward. Thus no real resistance is encountered up to the moment when the tube penetrates between the plateau of the cricoid cartilage and the posterior wall of the pharynx. At this point the posterior wall pushed ahead by the end of the tube looks like a collar.

At this instant the introduction of the tube encounters more resistance for, on the one hand, more pressure is required to displace the cricoid cartilage and, on the other hand, the cricopharyngeus muscle begins to contract. The pressure required to push the instrument through the cricoid isthmus varies greatly from one patient to another. It depends partly upon anatomical conditions (the upper teeth, a thick tongue, or a short neck), but above all upon the degree of contraction of the muscles of the neck and the pharynx. Once the tube has entered the cricopharyngeal pinchcock it is impossible to know if the midline is being followed or if, having deviated to one side, the lateral wall is being pushed upon, because in looking through the tube only the uniform appearance of the pharyngeal mucosa can be seen, without any detail.

Deviation is frequent, for the tube as it lies upon the convex anterior surfaces of the cervical vertebrae has a tendency to diverge from the midline. In order to keep the instrument as close to the midline as possible, it is helpful to follow the position of the neck, making sure that the tube lies in the same axis. If it appears that the tube has deviated from the midposition, it should be drawn back a short distance and then reintroduced in a direction thought to be

more nearly correct. While the tube is being advanced cautiously, the mucosa of the posterior wall of the esophagus becomes progressively paler in appearance. Finally the lumen of the esophagus appears as a whitish surface almost like that of a cicatricial constriction. This pallor is the result of the local ischemia provoked by the pressure made by the end of the esophagoscope against the posterior wall of the esophagus. Beginners often mistake this pallor for a pathological change in the mucosa and above all for a cicatricial condition of the wall of the organ.

Resistance to the introduction of the instrument is greatest precisely at this spot and it is often necessary to wait an appreciable period of time for the contraction of the cricopharyngeal muscle to relax. At this moment the tube must be held between the thumb and index finger to avoid a too precipitous plunge of the instrument into the lower esophagus when the contraction gives way. It is in this part of the procedure involving the passage by the cricoid isthmus that the benefits of a previous injection of morphine and atropine are observed, since this medication tends to diminish the intensity of the muscle spasm.

If the tube encounters unusual resistance one must not be tempted to employ force but should rather wait until the contraction gives way progressively ahead of a moderate degree of pressure by the instrument. Many times the patient makes a swallowing movement either spontaneously or on request and the tube will then slip readily past the cricoid constriction into the depths of the esophagus.

Spasm of the mouth of the esophagus is a common enough occurrence in the absence of any abnormality, but in the presence of a foreign body lodged in the lower esophagus it is often greatly accentuated. It is necessary, also, to distinguish the resistance caused by a true muscle spasm from that experienced when the end of the tube impinges upon the superior margin of the cricoid cartilage (Fig. 65). As the picture shows, this is most likely to happen when the head and neck of the patient are too much extended, as for example when the head of the patient in the recumbent position is allowed to fall too far below

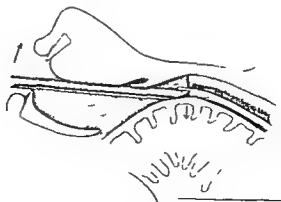


FIGURE 65 : As a result of excessive dorsiflexion of the vertebral column the esophagoscope often impinges upon the upper edge of the cricoid cartilage.

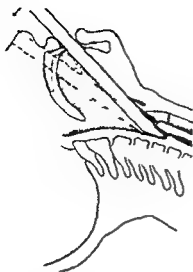


FIGURE 66 The head is not extended sufficiently and the tube impinges upon the posterior wall of the hypopharynx

the level of the examining table. In this position not only is the larynx forcibly pressed against the anterior surfaces of the vertebrae, but also the distal end of the esophagoscope, after having pushed the arytenoid cartilages forward, tends to move towards the superior edge of the cricoid cartilage. Obviously this resistance, often mistaken for spasm, does not disappear after the usual waiting period. If the operator allows himself to push harder, he may cause lacerations of the mucous membrane overlying the cricoid cartilage by the application of too much pressure. In some instances the examiner, after having perforated the mucosa over the superior margin of the cricoid, has been known to advance the tube between the posterior surface of the arytenoid and the mucosa to the point where bleeding makes it obvious that the tube is no longer within the lumen of the esophagus.

It is easy to avoid this second cause of resistance by making certain that the head and cervical spine are aligned in the correct position as described previously. The distal end of the instrument then avoids the cricoid cartilage and, aided by a correct position, slides into the deep part of the esophagus without resistance.

A third and important possibility of resistance encountered in attempts to pass the cricoid constriction is that produced by too sharp an inclination of the tube in the direction of the posterior wall of the pharynx. This unfavorable position of the instrument is produced by insufficient extension of the patient's head. It is particularly dangerous because the edge of the instrument pushes up a little jutting mound or collar of mucous membrane on the posterior wall against which slight additional pressure may cause a break in the mucosa or even a perforation of the wall (Fig. 66). To avoid this resistance and the danger of a serious injury, it is necessary at the moment when the cricoid constriction is to be passed for the assistant to hold the head in maximal extension so that the tube becomes parallel with the posterior wall of the pharynx and esophagus at that point (Fig. 66, dotted lines).

If the tube is pushed a little beyond the cricoid constriction the lumen of the esophagus appears toward the superior edge of the end of the tube while the posterior portion of the field of vision is occupied by the posterior wall of

the esophagus (Fig 67, insert) As the posterior wall is attached to the anterior surface of the vertebral column merely by a layer of loose connective tissue the distal end of the instrument tends to produce horizontal folds across its surface The air caught within the esophagus now begins to escape with a noise like eruption Often the air escapes before the lumen of the esophagus is widely opened

If, in spite of a correct position of the head and after a suitable delay, the passage of the cricoid constriction appears to be impossible, the following procedure may be employed The esophagoscope is removed the patient is made to sit up and a fine bougie, not over 3 to 4 mm in diameter, is introduced into the esophagus The patient is then requested to lie down again and the instrument is reintroduced according to the usual procedure as far as the posterior wall of the larynx (Fig 68) The bougie is found and the end of the

FIGURE 67 The tube has passed through the cricoid constriction The esophageal lumen half open is seen in the endoscopic view in the insert

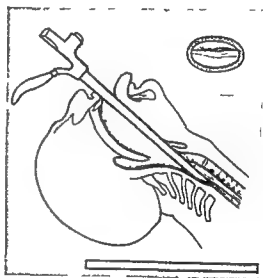


FIGURE 68 Passage of the cricoid constriction with the aid of a bougie



FIGURE 69 Endoscopic view during the passage of the cricoid constriction using a bougie as a guide



esophagoscope is kept in contact with it. In this manner the slight groove made along the posterior wall by the presence of the bougie as the end of the esophagus lies upon it is used as a guide, and the tube slides along the bougie across the cricoid narrowing and into the esophagus without encountering any appreciable resistance (Fig. 69).

On the average the distance between the upper dental arch (incisor teeth) and the cricoid constriction in an adult man is from 14 to 17 cm. and in a woman from 13 to 15 cm. Naturally, these figures are subject to considerable variation. They are of considerably more importance in adults than in children. Even in persons of equal height, this distance may vary because the cricoid cartilage may be relatively high or low depending upon the length of the neck. In order to reach the opening into the lumen of the esophagus, it may be necessary to push the end of the tube 20 to 23 cm. from the dental arch. Moreover the endoscopist must realize that not only does the distance between the cricoid constriction and the dental arch vary greatly, but also the position of the cricoid cartilage (and therefore of the cricoid constriction) is very variable in relation to the superior strait of the thorax. With a long, thin neck this distance between the inferior margin of this cartilage, which corresponds with the location of the mouth of the esophagus, and the upper edge of the sternum is from 5 to 6 cm., whereas in a person with a short neck the cricoid may be situated so low that its inferior border lies in the same plane as the upper sternal edge. In patients with such a low-lying larynx not only is the major portion or even the entire trachea located within the thorax, but also the corresponding segment of esophagus which would ordinarily be in the neck is endothoracic. Thus, in certain subjects after the cricoid constriction has been passed the esophagoscope instead of entering a cervical portion of esophagus enters directly into the thoracic segment of the organ.

This knowledge is important in regard to the therapy and prognosis of pathological conditions in the upper esophageal segment particularly in the case of perforations made by pointed foreign bodies or by the end of the esophagoscope. In patients with a larynx situated high in the neck, the lesion can be exteriorized, thus avoiding a descent of infection into the thorax. With a low-lying larynx the perforation is almost always intrathoracic and the result of surgical intervention is less favorable.

Once the barrier of the cricoid constriction has been passed and the lumen of the esophagus is opened, the most difficult and most dangerous part



FIGURE 70 The dorsiflexion of the cervical vertebral column here is too extreme and the anterior wall of the esophagus pressed upon by the tube pushes forward the posterior wall of the trachea.

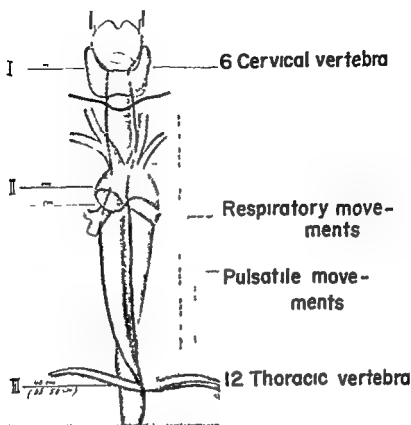


FIGURE 71 Diagram of the three physiological constrictions of the esophagus I the cricoid constriction II the aortic constriction III the diaphragmatic constriction (Haslinger)

of the esophagoscopy examination is over. The operator now must watch carefully to be sure that the instrument follows the axis of the esophagus, avoiding contacts of the end of the tube with the wall. If the end should begin to impinge upon the lateral wall of the organ, it is necessary merely to make an appropriate adjustment of its direction to put it back in the axis of the esophagus again.

If the esophagoscope becomes too much deviated towards the anterior wall, the head of the patient should be raised a little (Fig. 70). This movement lowers the end of the instrument. Too much pressure against the anterior wall obviously results in compression of the soft posterior wall of the trachea. To obviate this it is necessary at the time of introduction of the tube to avoid dorsiflexion of the cervical spine. The correct position, as emphasized before, is with the cervical spine anteflexed and the head in maximal extension.

With the distal end of the esophagoscope in the thoracic segment, the characteristic movements of the walls of the esophagus can be observed. These are the result partly of the effects of respiration and partly of the pulsations of the heart and great vessels (Fig. 71).

RESPIRATORY MOVEMENTS OF THE ESOPHAGUS During the examination of the thoracic portion, three different motions which depend upon the respirations can be distinguished:

1. The most noticeable movement of this source is characterized by varia-

tions in the size of the lumen of the organ during the phases of respiration. To understand this phenomenon it is necessary to recall that during inspiration there is an increase in the negative pressure within the thorax. The interior of the esophagus, because it communicates by way of the lumen of the esophagoscope with the outside air, has a pressure equivalent to that of the atmosphere. During inspiration, therefore, the interaction to the increased negative pressure of the thorax and the atmospheric pressure in the lumen of the esophagus causes the walls of the organ to distend. Thus during inspiration there is an absolute increase in the diameter of the esophagus. During expiration the reverse of this occurs. The intrathoracic pressure increases above that of the intraluminal air of the esophagus and compresses its walls together, thus bringing about a diminution in the diameter of the lumen.

These motions become much more noticeable during forceful respiration or bouts of coughing. During the expiratory phase of a paroxysm of coughing the walls are completely collapsed and the lumen of the esophagus disappears.

The respiratory movements begin to be observed after the passage of the cricoid constriction and are perceptible all the way to the cardia. The variations in intraluminal diameter described above are important because they indicate that the mucosa is normally flexible. In the event of an infiltration by a tumor, for example, the variations of the lumen cease because the thickening causes a rigidity of the wall so that it can no longer follow the respiratory movements.

2 The second type of motion associated with respiration is a sidewise swaying of the esophagus manifested by a slight forward movement during inspiration and a backwards recoil during expiration.

3 The third form of motion is an up and down movement imperceptible to the eye because it takes place in the long axis of the organ and undergoes a tremendous foreshortening due to the effects of perspective. This can be demonstrated easily by introducing a rather long pledget of cotton down the esophagoscope into the lower portion of the esophagus and inviting the patient to breathe deeply. It is obvious then that during inspiration the pledget descends 1 or 2 cm. and rises an equivalent distance during expiration.

Besides these motions of the esophagus induced by respiration, movements which are synchronous with the pulse can be seen. They are transmitted to the walls of the organ by the aortic arch, the descending aorta, and by the heart. Frequently they are not only visible but can be detected by the hand of the operator in the form of small rhythmic jerks of the instrument. These pulsatile movements may be observed from the level of the aortic arch to the cardia.

Even in the normal state the lumen of the esophagus presents an irregular shape which changes according to the segment which is under observation. Sometimes the perimeter of the view through the instrument is more or less round, again at other levels it is irregular. This variability of the lumen is due to the pressure exerted by adjacent organs and structures such as the trachea, the thyroid gland, the aorta, and the vertebral column upon the esophagus, the walls of which are soft by comparison. The most constant and obvious impression is that made upon the left side of the organ by the aortic arch (Fig. 72). This is more or less pronounced, depending upon the individual subject, but the walls are never compressed to the point of obliteration of the passageway.

It is always more obvious in adults than in children. From the physiological and pathological viewpoints, this narrowing does not have the importance of either the cricoid constriction or that in the region of the diaphragm. It is rarely necessary at this point to separate the walls with the end of the instrument as is always the case with the other two of the three narrow points of the esophagus.

Normally, as far down as the aortic arch the esophagus is relatively straight (Fig. 72). Beyond this point it presents a slight curve to the left and forward. This anatomical fact is most important when it comes to the examination of this region. If the esophagoscope is pushed a little further in the same direction that is employed in the segment from the cricoid constriction to the aortic arch, at a point about 23 cm. from the upper dental arch it will be seen that it is chiefly the posterior wall of the esophagus which occupies the field of vision and the lumen deviates upward (Fig. 73 insert). To avoid injuring the posterior wall while pushing the tube down and to keep it in the center of the passageway the head of the patient must be lowered and with it of course the instrument itself (Fig. 74). By this maneuver the distal extremity of the tube is elevated and put in line with the posterior esophageal wall. Since the usual Haslinger instrument reaches only as far as the region of the aortic arch, the inner elongating tube must be inserted at this stage of the procedure in order to examine the lower portion of the organ. This should be done before the head is lowered. With the Jackson esophagoscope of course, and with the newly modified Haslinger tube no additional apparatus is needed.

After the aortic narrowing has been passed, orientation becomes a little more difficult because the field of vision in the esophagus is smaller due to the

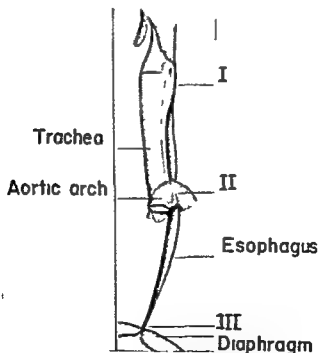


FIGURE 72 Lateral view of esophagus showing the three physiological constrictions: I cricoid constriction, II aortic constriction, III diaphragmatic constriction.

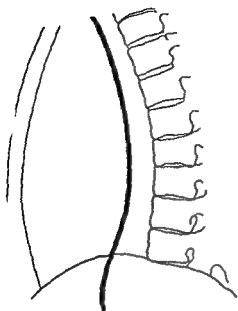


FIGURE 76

FIGURE 76 Position of the esophageal sound in a subject with a normal vertebral column (drawn from a roentgen film)

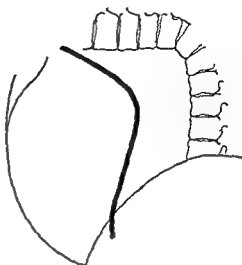


FIGURE 77

FIGURE 77 Position of the esophageal sound in a person with marked kyphosis (drawn from a roentgen film)

the folds described above will be seen, whereas in the latter the mucosa appears uniformly smooth

Furthermore, it cannot be argued that measurement of the distance the tube has been inserted by reading the figure at the level of the upper teeth is an accurate guide. Although 40 cm is an average figure for the distance between these teeth and the cardia, there are striking variations within the limits of normal. Actually in an adult man the distance may vary from 41 to 45 cm, and in an adult woman from 37 to 40 cm. In addition, these relations may be reversed, as for example in a small man the distance may be only 35 to 36 cm, while in an unusually tall woman it may be as great as 45 to 47 cm. Obviously in children these measurements are proportionately smaller. In a child of four years, for example, the distance from the upper teeth to the cardia is about 25 cm. It is clear, therefore, that as a guide to the progress of the instrument reading the figures marked upon the side of the tube is exceedingly unreliable. Actually, in order to know whether or not the cardia has been reached it is better not to rely upon the figures but rather to be guided by the characteristic esophagoscopic appearances described above.

It should be pointed out that the examination of the lower segment of the esophagus is more difficult and more uncomfortable for the patient whose body is heavy set and the neck short and stocky or when the upper teeth are unusually long than for the individual whose neck is long and thin or who has no teeth on the upper jaw.

The examination as a whole is exceptionally difficult in patients with a pronounced kyphosis or a kyphoscoliosis of the spine. Whereas in normal persons the esophagus assumes a slight anterior curvature below the aortic constriction, in kyphotics, by contrast, it often makes a sharp right angle curve

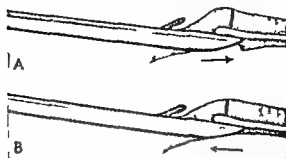


FIGURE 78 *A* During its introduction the end of the tube produces folds *B* during its withdrawal the tube smooths them out

These differences are made obvious by comparing Figures 76 and 77, which are diagrams made from roentgen films taken after the introduction of a radiopaque flexible bougie. Figure 77 makes it easy to understand that in a patient with severe kyphosis the examination of the lower portion of the esophagus may be very difficult or even impossible. It is obvious also, that in such a patient there is great danger of producing an injury or even a perforation of the lower esophagus, and greater caution must be exercised than when the spine is normal and the esophagus therefore lies in the usually expected position.

As the esophagoscope is withdrawn at the completion of the examination, the operator should pay strict attention to the direction of the esophagus. If an instrument with an elongating tube has been used, the latter must be withdrawn first. The retrograde examination, made during withdrawal of the instrument, is of great importance in visualizing certain areas especially the cricoid constriction because the visibility is actually better at the removal of the tube than during its introduction. As it is being inserted, the end of the instrument causes the mucosa to heap up in folds before it, whereas during its withdrawal the tube effaces these formations and often because of this a foreign body not observed during the introduction of the instrument may be disclosed (Fig. 78 *A, B*).

Technique with the Chevalier Jackson Esophagoscope

The esophagoscope evolved by Chevalier Jackson is a hollow tube of brass without an obturator and with no lateral openings. The proximal or ocular end is provided with a handle placed at a right angle which in normal use is held vertically upwards. The distal extremity is beveled (Figs. 79 and 80). In the wall of the tube are two channels which lie opposite each other. One of these has in its distal extremity a little recess. Into the lumen of this channel is thrust a rod at the end of which is an electric bulb. The length of this light carrier is such that when it has been inserted to the hilt, the bulb lies in the recess provided for it at the end of the instrument. The other channel opens in the distal extremity of the tube but not as far down as the electric bulb. Its proximal end can be attached to the aspirating apparatus to provide suction for the removal of liquids which might obstruct the view.

Thus the Jackson apparatus employs distal lighting; it must be introduced and manipulated under direct visual control and it provides a means of drying the endoscopic field.

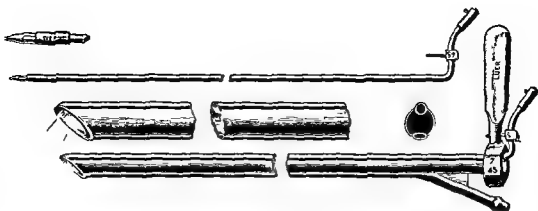


FIGURE 79 Chevalier Jackson esophagoscope with aspirator tube in the lower side

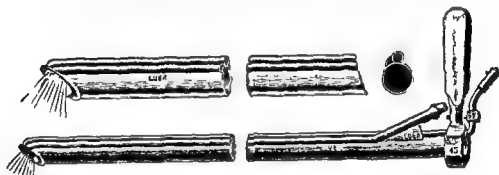


FIGURE 80 Chevalier Jackson esophagoscope with unimpeded lumen Aspirator tube on the outside

The following tubes are used in routine work.

For children under 3 years of age	5-6 mm × 35 cm
For adolescents	7 mm × 45 cm
For adults	9 mm × 53 cm

For certain purposes other tubes are employed

For the passage of large esophageal bougies, a model in which the light carrier and the aspiration channel are outside the lumen of the tube is employed. This location of the light carrier and aspirating tubes leaves the entire lumen free for the passage of instruments. This instrument is 9 mm by 45 cm (Fig 80).

Another special instrument of the Jackson type is the *esophageal speculum* employed for the examination of the upper third of the esophagus. There are two models, one for children and one for adults. These tubes are not graduated. This is a very practical instrument. Figure 81 illustrates the several models of this type.

The position of the patient is of great importance and demands the assistance of trained attendants. The patient should lie on his back with head and shoulders from the middle of the scapulae extending beyond the end of the table. The first assistant, seated on a stool to the right of the patient's head, places his left foot on a footrest the height of which has been chosen so that his left elbow can rest upon the anterior surface of his left thigh for support. His left hand can then support the occiput of the patient so that he can manipulate the head with ease in order to put it into the correct position. Then, passing his

right arm beneath the neck of the patient, he slips the right middle finger, armed with a mouth gag such as that depicted in Figure 82 into the mouth. Figure 85 shows the position of the patient.

In order to align the buccal cavity, the pharynx, and the mouth of the esophagus in the position indispensable for the introduction of a straight rigid tube, it is essential that the head be elevated approximately 15 cm above the horizontal plane of the table and that it be bent backwards at the atlo-occipital articulation in hyperextension. This double motion is most important because it places the cervical portion of the vertebral column in the same axis as the first portion of the thoracic spine. An appreciation of the value of this maneuver can be gained by comparing the roentgen film of a patient incorrectly placed (Fig 83) with that of a patient in the position described (Fig 84).

A second assistant helps to keep the patient flat on the table by pressing upon the anterior surfaces of both shoulders.

Finally, a third assistant holds the wrists and fixes the knees by resting his forearm upon the anterior surfaces of the patient's thighs. Figure 85 shows the position of the patient as held by the three assistants according to the recommendation of Dr. Chevalier Jackson for the use of the Jackson type esophagoscope.

Chevalier Jackson's technique demands that the esophagoscope must be introduced under direct vision and that the downward passage of the instrument must likewise be controlled visually. The operator standing erect grasps the distal end of the tube between the thumb and index finger of his left hand and the proximal extremity at the collar (not by the handle) with the fingers of his right hand as if he were holding a pen. He then introduces it vertically along the tongue as far as the posterior wall of the pharynx, holding the instrument in

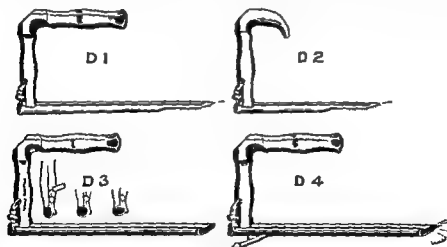


FIGURE 81 Esophageal speculum Chevalier Jackson type showing different models

FIGURE 82 Mouth gag bent at an obtuse angle (Soulas)





FIGURE 83 Roentgen view showing incorrect position for esophagoscopy. The vertebral column is not straight but maintains its normal curve.



FIGURE 84 Roentgen view showing the correct position with regard to both head and neck. The cervical vertebral column is straight (Chevalier Jackson).

the midline of the mouth with the handle uppermost (Fig. 86). During this maneuver it is indispensable to have the tube held against the arch of the upper teeth by the free fingers of the left hand, which are employed to prevent the upper lip from being pinched between the teeth and the instrument.

Once the posterior wall of the pharynx is reached, the following steps must be observed:

1. First, by making upward pressure with the left thumb, the end of the instrument is raised into the position to disclose the right arytenoid. Once this



FIGURE 85 Position of the patient and the attendants for esophagoscopy according to the Chevalier Jackson technique



FIGURE 86 Introduction of the esophagoscope - Chevalier Jackson technique



FIGURE 87 Passage through the mouth of the esophagus under visual control the assistant standing at the shoulders points out the suprasternal notch Chevalier Jackson technique



FIGURE 88 Exploration of the lower third of the esophagus Chevalier Jackson technique

landmark has been recognized with the mouth of the instrument still raised the tube is slipped into the right pyriform sinus

2 The second step, which involves the passage through the cricopharyngeal pinchcock, is of crucial importance. With the handle kept constantly uppermost, the upward motion being maintained by pressure of the left thumb the esophagoscope still held in the midline is threaded towards the suprasternal notch. This landmark is helpful in following the correct direction. Firm, continuous pressure is then exerted to cause the cricopharyngeal sphincter to relax. This is done as for any sphincter by maintaining a sustained gentle pressure without the exertion of any force. The sphincter opens in the upper part of the endoscopic field and, as this occurs the tube is inserted into the lumen still under direct visual control. As this is done the cricopharyngeal fold which corresponds to the lip of the mouth of the esophagus is passed and the esophagus is entered (Fig 87).

3 The third step comprises the exploration of the lower esophagus which does not vary from that described previously for the Haslinger instrument (pages 95-98). At this point the head of the patient should be lowered in such a manner as to bring the lumen of the esophagus into alignment with the axis of the lumen of the instrument (Fig 88).

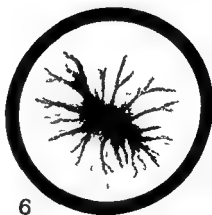
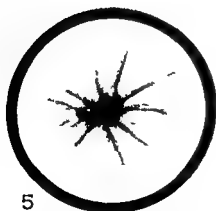
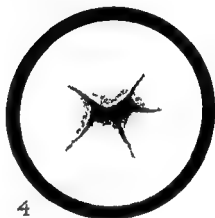
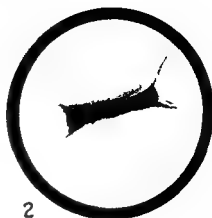
4 The fourth step involves the passage of the instrument through the hiatus in the diaphragm and into the cardia. With the head and shoulders held on a plane slightly lower than that of the table the assistant bends the head and shoulders towards the right avoiding any twisting motion. The operator then points the tube in the direction of the anterior superior spine of the left ilium. Once the location of the hiatus has been recognized the tube is made to pass into the cardia by means of gentle firm pressure (See pages 98-99).

Retrograde Esophagoscopy

This procedure has been employed to overcome strictures which could not be dilated from above. A gastrostomy stoma is used for the introduction of the esophagoscope. The chief difficulty is the discovery of the cardia. Attempts to find it by the sense of touch may succeed. Some operators have advocated the use of a cystoscope with the stomach full of water but this is always inconvenient. Sometimes the instrument can be guided to the cardia along a small bougie if one can be passed successfully through the stricture. Recently the operation has been carried out with much greater success under the control of the fluoroscope.

The patient's stomach must be empty. He is placed upon the fluoroscopy table with a sandbag beneath the lower dorsal region in order to elevate the upper abdomen. His knees should be semiflexed. After removal of the gastrostomy tube the field is prepared and draped with sterile sheets. After the eyes have become accommodated to the dim light of the room the well-lubricated end of the esophagoscope is introduced into the gastrostomy opening. When its tip is felt to be free in the hollow of the stomach it is pushed 3 or 4 cm. in an upward and backward direction. The radiologist then starts his machine and on the screen the angle formed by the vertebral column and the left cupola of the diaphragm is identified. The end of the tube is pushed in the direction of

PLATE I



ENDOSCOPIC VIEWS OF THE NORMAL ESOPHAGUS

1 The mouth of the esophagus 2 upper cervical segment 3 midcervical segment
4 thoracic segment 5 lower thoracic segment just above the diaphragm 6 cardia

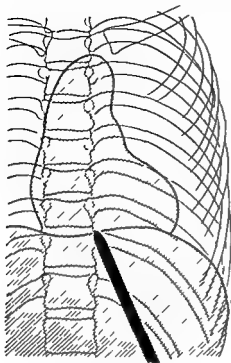


FIGURE 89

FIGURE 89 Retrograde passage of the cardia under fluoroscopic control (Technique of A. Aubin in Thesis of Torre)

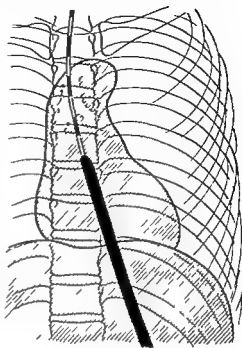


FIGURE 90

FIGURE 90 Retrograde passage of a bougie through a stenosed esophagus by way of the cardia (Technique of A. Aubin in Thesis of Torre)

this angle until the presence of the vertebral column can be felt (Fig. 89). With the end of the tube placed along the left side of the spine, gentle sidewise motions are made without any backward thrust and without lowering the tube too much when all of a sudden the end of the instrument appears behind the heart. This means that the esophagus has been entered. Once this has been accomplished, radiopaque dilators or sounds can be passed through the esophagoscope into the stricture under fluoroscopic control (Fig. 90).

This method is excellent because of its simplicity and relative safety. It is useful in the treatment of stenoses which seem to be impervious by the peroral route. It easily permits the insertion of a filiform bougie. The only contraindication to its use is the presence of a lesion of the cardia itself, whether cicatricial stenosis, ulcer, or tumor. Its success depends, however, on having a simple gastrostomy stoma, not one such as the Spry, Jaboulay, or Beck-Jianu types which are made with a tunnel of gastric wall.

The Accidents of Esophagoscopy

Every year, even in the hands of the most experienced and skillful endoscopists who are well acquainted with the methods of examining the esophagus, serious or even mortal accidents occur. There are several reasons for this.

In previous pages it has been emphasized that the anatomy of this organ is subject to variations. All the measurements and the numerical averages given in classical treatises are approximate, especially when they have been based upon

observations of the cadaver. In the living subject the dimensions vary not only according to the height and sex, but also with the intrathoracic pressure, the relative emptiness or fullness of the organ, and the position of the subject. To avoid embarrassing surprises during esophagoscopy, as emphasized above, it is of enormous importance before the examination is begun to inspect the roentgen films of the esophagus made after the ingestion of an opaque substance.

The esophagus is not a rectilinear tube. Its two lateral curves, above to the left and below to the right, make little difference in the average subject. But if the vertebral column is deformed, the esophagus undergoes a change of direction and bends according to the necessities of its position within the chest. In other words, the esophagus adapts itself to the conditions which exist within the mediastinum.

Furthermore, the organ is characterized by its movability, including the peristaltic activity of its muscular coats which induces the progression of a bolus of swallowed material. Likewise, the loose periesophageal connective tissue and the pulmonary, tracheal, and aortic attachments are extremely lax. It is this movability and laxity which make perforation by the long rigid esophagoscope easy. Once there is some abnormal rigidity of the wall, however, the danger increases. Carcinoma, the most common disease of the esophagus, leads to such a situation.

Another matter of importance in the production of injuries to the esophagus is the presence of the sphincters. The one true sphincter lies at the mouth of the esophagus. At the lower end is the pseudo sphincter in the hiatus of the diaphragm. These two extremities of the esophagus are most often involved in pathological conditions. One must realize that there exists a sort of physiological synchronism between these two areas of constriction. As soon as the upper has been passed, barring any congenital or acquired abnormalities, the diaphragmatic portion opens. Furthermore, concomitant spasm of both extremities of the organ is of frequent occurrence.

Lastly, the extreme thinness of the wall of the esophagus is not conducive to the introduction of a long rigid tube without injury. Furthermore, the seriousness of a perforation is augmented because of the possibility of infection of the mediastinum by the mixed bacterial flora of the mouth which normally contaminates the interior of the organ.

The accidents usually encountered are wounds of the wall of the pharynx, of the arytenoids, or of the posterior surface of the cricoid and perforations of the esophagus.

Wounds of the wall of the pharynx are produced by the end of the esophagoscope when it is too sharp or when it is unskillfully manipulated. The same is true for injuries to the mucosal covering of the arytenoids and the mucosa overlying the cricoid which are thin and very mobile. All of these injuries, no matter how small they may be, are characteristically associated with the occurrence of subcutaneous emphysema. If the injury is slight and does not become infected, there are usually no sequelae. If, however, the wound is large and especially if it involves the arytenoid region, infection is the rule. The loose periesophageal connective tissue layer becomes involved and the patient may die as a result of the occurrence of a phlegmon in the neck or of mediastinitis.

A special chapter is devoted to the consideration of *perforation* of the esophagus from all causes (Chapter 23)

Prevention

The best treatment is to avoid these accidents by observing the details of the proper performance of the examination as described above. The most important point is the passage of the pharyngo-esophageal segment, which is the blind spot of all esophagoscopy. The anatomy of this region has been emphasized, particularly the weak areas. Not only are the circular muscle fibers at the mouth of the esophagus (the cricopharyngeus muscle) difficult to overcome, but also the interlacing of the fibers is not firm at all points. In fact, there are interstices not only between the oblique fibers and the circular fibers of the inferior constrictor muscle, but also between the bundles of the cricopharyngeus muscle itself. Therefore, for the same reason that the mucosa may become herniated through this weak point to form a pulsion diverticulum of the Zenker type, an unskillfully managed esophagoscope pushed too forcibly or without visual control may become caught in this weak area, push the mucous membrane ahead of it, and then perforate the wall (Fig 91).

This leads to two important observations made by Chevalier Jackson, the first of which is that if the esophagoscope is allowed to follow the direction it would normally take and is pushed in forcibly it will certainly perforate this weak point into which it is naturally guided by the surrounding structures. The second is that it is important to combat the tendency, with the subject in the recumbent position to push the open end of the tube forward in the midline.

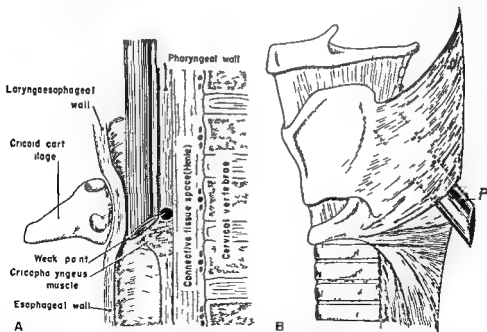


FIGURE 91 Mechanism of instrumental perforation of the esophagus. The end of the tube impinges upon the cricopharyngeus muscle. *A* If this muscle contracts and the tube is pushed posteriorly the end of the instrument rests upon the upper weak point. *B* If the operator exerts any pressure the tube perforates this weak spot.

because of the smaller caliber of the organ, and a fatal outcome may be the result

In attempts to secure biopsies from the abdominal segment of the esophagus or the cardia there is likewise great danger of perforation with its attendant complications. Here, because of the uncertainties of the roentgen diagnosis, the endoscopist is often asked to perform a biopsy, but because of the depth at which it is necessary to work and the great length of the biopsy forceps which must be employed, the greatest prudence must be observed.

5 A biopsy *should not be attempted in complicated or advanced cases of carcinoma*, such as those with recurrent nerve paralysis or with tracheo-esophageal fistulae. In these cases the growth has spread beyond the esophagus with invasion of the mediastinum and it is easy to penetrate beyond the protective barriers.

6 One *should not hesitate to repeat an esophageal biopsy*. As is true of every tubular organ, the edges of the lesion are often bounded by inflammatory tissue and if the bite of the forceps, because of the difficulty of working from above downwards, is not quite deep enough, the report from the laboratory may read "chronic inflammation." If there is clinical or roentgen evidence of a probable carcinoma, another attempt should be made and a deeper bite of tissue obtained. However, the experienced endoscopist can usually tell by gross examination if a satisfactory specimen has been obtained. The difficulties experienced in biopsies of the esophagus are analogous to those in the rectum and sigmoid.

Because a biopsy of the esophagus is always a relatively dangerous procedure, it should be attempted only when the findings might influence the surgeon against the performance of an operation, as in the case of a suspicious looking roentgen shadow in an elderly person in whom an operation would present a serious risk. In the majority of instances the roentgen appearance of the lesion is sufficiently suggestive to make the decision, and clinical judgment based upon experience usually leads to the correct decision.

A further word of caution is to refrain always from attempts to obtain biopsy material from any intramural tumor covered by normal-appearing mucous membrane. These are almost always leiomyomata or more rarely cysts, which can be enucleated through the outer wall of the esophagus without opening the lumen. If a previous biopsy has been attempted the injured mucosa may present difficulties for the surgeon when he performs the removal of the growth (see Chapter 26).

Good results have sometimes been obtained by examining bits of tissue rubbed off a lesion of the esophagus on a bit of cotton or gauze fastened to the end of a long applicator. Sometimes these fragments are just as good as those obtained with a biting forceps. If strands of tissue are found after careful inspection of the applicator upon its withdrawal, they should be immersed in 20 per cent formalin solution immediately. Some of these specimens may even be large enough to make paraffin sections.

Exfoliative Cytology

If a large piece of tissue is not obtained, the end of the applicator is rubbed across a glass slide for a smear which can then be examined by the Papanicolaou

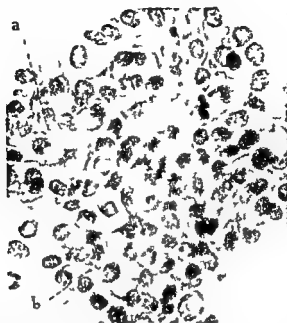


FIGURE 92 Photomicrograph of a shred of mucous membrane of the normal esophagus obtained from wipings with a swab. Note in *a* and *b* the cohesion of the epithelial cells and the sharpness of the cytoplasmic limits. $\times 565$ (A. Herbaut.)



FIGURE 93 Photomicrograph of a group of malignant epithelial cells from a carcinoma removed by wiping with a swab. The disorder of the cells is striking as well as the irregularity of their nuclei.

technique for evidence of malignant cells. Fixation of the smear with absolute alcohol and staining with hematoxylin and eosin by the usual techniques may produce satisfactory results as well. For comparison, Figure 92 shows a shred of normal mucosa and Figure 93 a bit of carcinomatous tissue obtained in this manner.

Recently, great assistance has been obtained by *cytological studies of saline*

washings obtained from the lumen of the esophagus above the suspicious lesion using the method of Papanicolaou. At the Massachusetts General Hospital it has been possible to identify positive malignant cells in 98 per cent of the patients with proven carcinomata of the esophagus. In a significant number of these patients malignant tissue had not been found in biopsy specimens removed through the esophagoscope by the usual technique. In several instances, furthermore, a positive diagnosis was obtained in patients with presumably benign disorders, such as achalasia, with subsequent proof by finding a carcinoma at operation. With the benefit of this additional technique, therefore, relatively few biopsies of the esophagus need be attempted, and the patient may thereby be spared the dangers and discomforts of the passage of an esophagoscope.

CHAPTER 5

Instrumental Exploration (Bougienage)

FOR MANY years before the advent of roentgenology and the esophagoscope the older authors were forced to emphasize the use of sounds to explore the interior of the esophagus. Except for their clinical knowledge which was sharpened by the difficulty, our ancestors had nothing but the sound and examination at necropsy to learn about esophageal disease and when the one led to the other, they could not truly be blamed. Mouton, writing in 1874, said, 'The use of the sound is a safe procedure providing the nature of the pathology which is under investigation is taken into account.' But he added 'How dangerous this maneuver can be when carried out before any other examination of the patient as the principal method of establishing a diagnosis!' In the present day, although some experts still advise the use of an exploratory sound or bougie with which to palpate the interior of the esophagus, others who are usually endoscopists of the greatest authority, such as Chevalier Jackson, interdict this method of probing in the dark. From the diagnostic angle therefore the sound is usually employed as an adjunct to esophagoscopy and under direct visual control as described previously (Chapter 4).

On the other hand, in the treatment of certain diseases of the esophagus, the passage of a bougie or sound plays a very important role. Sometimes its use is prophylactic, as in acute corrosive esophagitis to prevent the development of strictures. At other times it is employed as a method of treatment to dilate stenoses, in certain spastic conditions, and as palliation occasionally in cases of carcinoma. Rarely it may play a part in the radiation therapy of neoplastic disease.

We shall consider the use of the method therefore, first for exploration under visual control and second as a part of therapy (see Chapter 19).

Bougienage as a Method of Exploration

As a method of palpation, the use of the exploratory bougie is the equivalent of elongating the finger in order to employ the sense of touch. By this means a better appreciation of the relative rigidity or flexibility of the esophagus in any given area can be obtained than by using the esophagoscope alone. This is valuable in the study of the friability of a tumor, the thickness of a given portion of tissue, or the resistance of a spastic area. Likewise, in the case of large impacted foreign bodies it is sometimes helpful to make a preliminary examination with a bougie in order to estimate the location of the object and its distance from the mouth. The instrument must, however, be handled with great care and manipulated gently. Sometimes by this means the foreign body may be disengaged or even free spaces may be created beside it which facilitate grasping it with an extracting forceps. In some instances a small bougie may be employed to test the presence of a lumen through a carcinoma for the passage of a larger sound, or, in cases of stricture from chemical burns, to establish the passageway under visual control before passing the larger dilators.

General Considerations

The use of the bougie should always be preceded by an examination of the patient, particularly inspection of the neck and chest, review of the roentgen films, and determination of the body temperature, elevation of which would contraindicate the performance of any manipulation in the esophagus.

The first passage of the bougie in any case must be with visual control through the esophagoscope. Later, during the course of a dilatation through an



FIGURE 94 The consequences of blind bougienage (After Chevalier Jackson)



FIGURE 95 Cylindroconical bougies of soft gummed silk with olivary tips (three sizes)



FIGURE 96 Chevalier Jackson bougie for esophagoscopy dilatation

area which is not angulated or otherwise in danger of perforation the experienced surgeon (preferably a specialist in endoscopy) who has in his mind a clear picture of the configuration of the organ will be able to pass the bougie without the assistance of the esophagoscope

It must be re-emphasized, however, that blind bougienage is often useless and always dangerous. An example of the dangers involved can be appreciated by the study of Figure 94, which shows the specimen removed at autopsy following an instrumental perforation with subsequent periesophageal abscess formation.

The most frequently used bougie is made of woven silk coated with gum. A rubber model is also available. The ordinary bougie is invisible under the fluoroscope. In order to overcome this inconvenience the interior is sometimes filled with mercury or lead shot. Actually when opacity of the instrument is desired as in the exploration of certain strictures the bougie is usually filled with a substance containing bismuth or barium. The tapered bougies of gummed silk recommended by Guisez and Jackson are illustrated in Figures 95 and 96. All of these instruments are made of the material used for many years by urologists. In fact, in children who have a high lying stricture the use of ureteral bougies to explore the lesion has been recommended by some authors. In persons who have a gastrostomy this instrument may be employed under the fluoroscope to assist in the passage of a thread for subsequent guidance during dilatation.

Contrary to that of the urological instruments however, the measuring scale on the esophageal bougie is graduated in one-third millimeter instead of one-eighth millimeter.

It is important to emphasize that for instrumentation with a bougie whether exploratory or therapeutic, nothing but the soft waxed cylindrical type with a conical olivary tip should be used.

Technique

So far as the actual technique is concerned, a careful description is given because with patients who require repeated dilatations over the course of many years, as in the cases of chronic stricture resulting from chemical burn it is often necessary for the family physician or local surgeon to take over the respon-

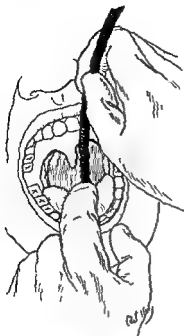


FIGURE 97 *Introduction of bougie*
First step The bougie, held in the right hand like a pen, is introduced at the back of the pharynx. Light pressure by the left index finger depresses and immobilizes the base of the tongue.

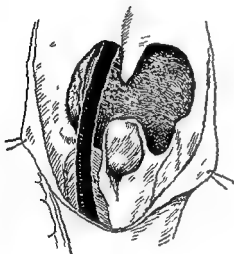


FIGURE 98 *Introduction of bougie*
Second step Passage of the bougie behind the larynx at the bottom of the junction of the larynx and pharynx. More exactly bougie must descend behind the arytenoids and exactly in the midline. In the drawing the bougie has been purposely shown at one side in order to avoid obscuring the orifice of the larynx (Viewed from behind as if pharynx were opened).

sibility for the treatment. Certain maneuvers are important and the risks are great, especially if the patient is uncooperative.

A sterilized bougie of No. 25 to 30 caliber should be chosen to start. The subject, after having removed any dental prostheses which may be present, should be seated facing the operator.

In the majority of instances no anesthesia is required. With an unusually timid patient it is wise to administer a preliminary sedative of the operator's choice. A 5 per cent solution of cocaine or one of its less toxic related topical anesthetics may be used as a spray. This is less uncomfortable for the patient than the curved cotton applicator. While this is being done, the patient's tongue is held forward with a piece of gauze in the hand of the operator or the patient. The patient should be warned of the temporary interference with deglutition produced by the anesthetic. Furthermore, in order to avoid the danger of any unexpected sudden movement on his part, the patient should be told to raise

his hand if he should experience any pain. The instrument can then be hastily withdrawn. The bougie is kept lubricated with sterile olive oil or glycerin.

The patient's head should be held vertically or bent slightly forward in order to overcome the sharp curve described by the vertebral column from the sixth cervical down to the third thoracic vertebra. If the head is bent too much, the larynx and trachea may be entered. The patient should breathe deeply with his mouth open. With his right hand the physician then grasps the bougie near its terminal one third, holding it as he would a pen, and with the base of the tongue depressed lightly by the left index finger and with the bougie slightly curved, he introduces the instrument deep into the pharynx (Figs 97 and 98). The two hands are henceforth used together. The right hand, with which the bougie is made to slide along the midline of the posterior wall of the pharynx, is supported lightly by the left, the index finger of which, lying constantly on the base of the tongue, is used to guide the bougie. In case the instrument is made to deviate sidewise from its correct position by an unexpected reflex due to nausea (gag reflex), the left index finger is used to guide it back to the midline again. The patient should be encouraged to breathe as quietly as possible. As the mouth of the esophagus is reached in this manner, a sphincteric contraction is often encountered (Fig 99). The patient is then asked to swallow and with gentle firm pressure the bougie quickly enters the cervical esophagus (Fig 100) and, if unobstructed, passes readily through the thoracic and diaphragmatic portions to reach the cardia. If at any point in its course the bougie meets with persistent resistance, it *must not be forced*. If the resistance is at the mouth of the esophagus, a second attempt may be made after anesthetizing that area in particular with a laryngeal cotton applicator slipped along the posterior wall of the esophagus.

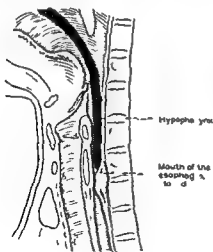


FIGURE 99

FIGURE 99 Introduction of bougie. Third step. The bougie after having traversed the hypopharynx encounters a normal resistance at the mouth of the esophagus which must not be breached forcibly. This resistance varies in degree from person to person.

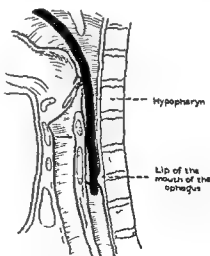


FIGURE 100

FIGURE 100 Introduction of bougie. Fourth step. Under gentle continuous pressure the mouth of the esophagus is passed by. From here the way is clear as far as the hiatus in the diaphragm.

Certain unimportant minor incidents may occur. The patient may cough, become excited, and appear to be asphyxiated. This occurs when, because of an instinctive movement of recoil from the instrument, the head becomes bent at the wrong angle and the tip of the bougie touches the mucous membrane of the larynx. This trouble is soon overcome by withdrawing the instrument and replacing the patient's head in the correct position for another trial.

Complications

The complications are chiefly perforation and the creation of false passages. These are the specters to be avoided. Acute esophagitis and the dilatation above a stricture are two conditions which lead to a dangerous increase in the friability of the esophageal wall. Cases of recent chemical burn and neoplasms provide the most frequent contraindication to blind exploration of the esophagus with a bougie, but the method is rarely employed in such cases.

Caution

The principal rule is never to employ force in the passage of a bougie. When after the body has been opened and one has seen laid out upon the autopsy table an esophagus previously damaged by caustics, when one examines carefully the alternating areas of thickening surrounded by bands of periesophagitis and the intervening segments, the mucosa of which is extremely thin, each connected with the other by eccentrically placed channels representing all that is left of the lumen, and when one observes the pockets of stasis or pseudo-diverticula, the danger of all blind exploratory bougienage in such cases becomes obvious. One can appreciate what harm such a maneuver can cause. What is more, one can measure the possible seriousness of an attempt at dilatation even though it is carried out according to all the rules, and one can foresee the fulminating complication which may carry off these patients in a matter of hours. One is reminded of the haunting, terrible dictum of Trousseau: *"Sooner or later these patients die killed by the bougie."*

The use of bougienage as a method of treatment is presented in Chapter 19.

CHAPTER 6

Congenital Malformations

CONGENITAL malformations of the esophagus are not uncommon. They may be limited to the esophagus itself or because of a common embryological origin, they may be accompanied by abnormalities of the trachea the bronchi or the thyroid or thymus gland. Some are incompatible with survival and lead to the death of the infant during the first few days after birth. Others though not lethal may be found accidentally in later years at autopsy or they may be serious enough to cause functional difficulties from the first. The recent advances in thoracic surgery have greatly altered the prognosis however and now many infants, particularly those born with congenital atresia can be saved with an excellent prospect of their leading a normal existence.

Review of Embryology

At the end of the first month of embryonic life the future intestinal tract is represented by a ventral gutter which extends from the cephalic to the caudal extremity and opens into the yolk sac. Ultimately by reason of its elongation and the formation of the cardiac and allantoidal folds, this gutter becomes transformed into a tube. While this tubular transformation takes place promptly at the cephalic and caudal ends, it is slower on the side at the yolk sac. In this manner three principal portions are formed. One is the cephalic region or foregut (the pro-enteron) the second is the midgut or abdominal intestine (the meso-enteron) and the third is the hindgut (or infra-enteron). The pharynx and the esophagus develop from the foregut (pro-enteron).

Situated in the median plane, this primordial intestine lies against the ventral surface of the neural tube. With the formation of the notochord the mesenchyma developing gradually around it elongates and brings about a separation between the intestinal wall and the neural tube. As this occurs short thick mesenchymatous masses reattach the pro-enteron to the posterior thoracic wall to form the mesentery.

Then on each lateral surface of the tube a longitudinal furrow appears ex

ternally, and in consequence a ridge appears internally along the lumen of the tube. These furrows gradually become more and more accentuated, and the two ridges on the inside come into contact and finally become joined to form a partition which brings about a reduplication of the foregut to form two tubes. The dorsal tube becomes the esophagus and the ventral becomes the respiratory tract. From the latter develop first the larynx and trachea and finally the lungs, which bud forth to look at first like two diverticula.

Above, however, at the upper or forward end of the cephalic intestine, this separation into two channels does not pass beyond the fifth cervical vertebra from which to the base of the skull there is only one tube which becomes the pharynx. This pharynx, consisting at first of a large ring of muscle which becomes attached to all the bony, cartilaginous, or fibrous surfaces available in the cervical region, adapts its structure to its ultimate function of propulsion of food into the alimentary tract and as an accessory purpose the conveyance of air into the respiratory passages.

In the beginning the esophagus is merely a very short muscular ring situated between the larynx and the stomach, all of which at that time lie in the neck. As development progresses, it becomes elongated following the enlargement of the neck and the formation of the thoracic cage and the lungs. It becomes attached to the vertebral column by the mesenchymatous layer called the dorsal mesentery and to the posterior surface of the heart by the ventral mesentery or posterior mesocardium.

Later, the part of the tube which forms the stomach undergoes a process of rotation and dilatation which produces a turn of 90 degrees on its long axis from the primitive midline or sagittal plane position, so that its posterior surface moves to the left and its anterior surface to the right. At the same time its anterior portion bulges to the left. These two motions of twisting and dilatation bring about a rotation of the lower portion of the esophagus in the same direction as that of the stomach.

The development of the spinal column and that of the diaphragm have their effects upon the esophagus also, as we shall see (page 146).

In general there are two types of deformity, one involving the esophagus only and the other the trachea as well.

Malformations of the Esophagus Alone

Six variations of this abnormality may be seen.

1 *Total Absence of the Esophagus* This is infrequent and is found usually only in monsters. It may exist alone or in association with malformations of other parts of the gastrointestinal tract or of the respiratory system.

2 *Partial Absence of the Esophagus* In this type of case the lower portion is missing and the proximal part ends in a cul-de-sac in the base of the neck or upper thorax. A similar cul-de-sac may exist at the cardia. It is an unusual anomaly (Fig. 101).

3 *Complete Absence of the Lumen* In this instance the esophagus throughout its entire length is nothing but a cordlike structure consisting of connective tissue with sometimes an admixture of muscle fibers (Fig. 102).

4 *Partial Absence of the Lumen* This may occur in the midsegment only, with an upper and a lower tubular portion joined by a fibrous cord, or it may exist in the lower portion with the proximal portion essentially normal though dilated. In the first of these the upper third of the esophagus is dilated, with a large lumen and a hypertrophied wall rich in muscular tissue. The lower third is by contrast very narrow. The cord which unites them is frequently short, so that occasionally an anastomosis can be performed if the condition is recognized promptly. The cord usually consists of connective and muscular tissues. Sometimes it is almost entirely muscular, sometimes mostly fibrous (Fig 103).

5 *Double Esophagus* Reduplication may occur in the esophagus, as in any other portion of the alimentary canal. It may involve the entire length or only a portion, but in either event the two branches are usually united at the lower end.

6 *Membranous Diaphragms and Congenital Strictures* These are the most frequently encountered malformations of the esophagus alone and actually the most amenable to surgical treatment. They may be weblike membranes seen either in the upper third or in the lower portion just above the cardia. Sometimes they involve a short length of the organ in a fibrous tissue stenosis or stricture resembling that produced by an inflammatory process (Fig 104, A, and B).

Very few of these malformations of the esophagus are compatible with survival. There are, however, patients who are born with congenital webs or



FIGURE 101

FIGURE 101 Absence of the esophagus. Nothing remains but two independent blind pouches, one at each end.



FIGURE 102

FIGURE 102 Absence of the esophageal lumen. The organ is replaced by a solid cord extending its entire length.

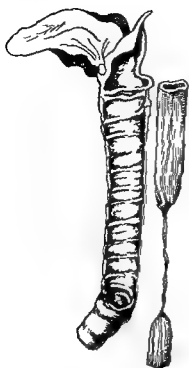
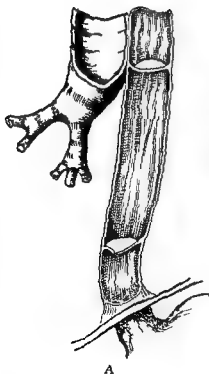


FIGURE 103 Partial absence of the esophageal lumen. A solid cord of variable length unites the proximal and distal blind pouches.



A



B

FIGURE 104 A Intraluminal congenital membranous diaphragms. The valve like arrangement is typical. B Esophagogram showing two congenital weblike strictures. child aged 4 years (Glass plate one of the first roentgen examinations of the esophagus ever made in Boston operation at age 54.)

stenoses which are only partially complete so that they may live, although the nature of the difficulty may not be discovered until later in life (Fig 104, B) Rarely also, mega-esophagus indistinguishable from achalasia in older patients may occur as a congenital disorder

Complete Stenosis

CLINICAL CHARACTERISTICS In some instances the stenosis is complete as a result of a membrane of more or less solid consistency located usually just above the level of the diaphragm and completely occluding the lumen of the esophagus When visualized through the esophagoscope it is shiny, of an iridescent hue or grayish red The infant is unable to swallow, becomes cyanotic, and then is relieved by regurgitation of the food it has attempted to get down Fluoroscopy demonstrates a stoppage of the contrast medium at the level of the membrane and absence of gas in the digestive tract below These observations serve to differentiate this condition from congenital atresia with tracheo esophageal fistula

TREATMENT The condition may occasionally be overcome by the passage of a bougie under the guidance of the fluoroscope Surgical intervention is, however, almost always the only effective method of treatment This may be postponed until the child has grown larger if a feeding gastrostomy is performed as a temporary expedient

Partial Stenosis

CLINICAL CHARACTERISTICS In certain cases the membranous stenosis is incomplete This type is more frequent than that with complete occlusion, but it is still rather uncommon among abnormalities of the esophagus Guisez reports the discovery of five cases in 2000 esophagoscopic examinations This type is characterized by an abnormal narrowing of a limited portion of the esophagus without any evidence of a pathological condition of the tissues at the level of the constriction These stenoses are usually found in the lower third of the esophagus almost always at the lower end Sometimes they may be found as high as the tracheal bifurcation and rarely at other levels Occasionally more than one may exist in the same patient The condition is usually recognized early in young infants of a year or more or in adolescents When it is recognized in later life careful questioning reveals the fact that the difficulty began at an early age (Fig 104 B)

In infants under one year of age the symptoms are not obvious and the true cause of the trouble often goes undiscovered The child does not develop properly, but the milk passes into its stomach Stools and urine are normal Often a gastric or intestinal affection is suspected There is often habitual vomiting

Depending upon the degree of narrowing which exists these troubles become manifest more or less early but in general the situation becomes obvious when a change from liquid to solid food is made As semisolid food is begun, the vomiting increases dysphagia sometimes becomes complete often rather suddenly, and the roentgen and esophagoscopic observations then made uncover the diagnosis Sometimes the physician or the mother returns to liquid feedings

DISEASES OF THE ESOPHAGUS

with relief and the discovery of the condition is thereby that much delayed. Sometimes the true nature of the trouble is disclosed only when complete obstruction develops as a result of the lodgement of a piece of meat or the seed or pit of some fruit. As a result of stasis in the esophagus above these constricting diaphragms, surface irritation of the mucosa may occur, leading sometimes to esophagitis and further cicatricial stenosis.

ROENTGEN EXAMINATION discloses a stenosis with fusiform proximal dilatation.

At ESOPHAGOSCOPY the esophagus is often found filled with old, partially decayed food, and after cleansing by wiping or lavage, a transverse diaphragm can be seen. There is usually a zone of constriction or a sort of web, often with two lips which tend to meet in the midline, the edges of which appear granular and inflamed. Sometimes the constriction looks almost like a sphincter, or like a purse drawn together with a string, in the center of which is a narrow aperture representing all that remains of the lumen at that point. Sometimes this opening is eccentric in relation to the circumference of the esophagus. These are the two types usually encountered. Sometimes the constriction appears to involve mostly the muscular coat without the presence of a web in the mucous membrane. These cases, however, are actually instances of congenital hypertrophy of the circular muscle fibers somewhat analogous to the situation in hypertrophy of the pyloric sphincter. They are, therefore, actually examples of congenital achalasia of Type 2 to be described in Chapter 9.

DIFFERENTIAL DIAGNOSIS In summary, a lower esophageal web of congenital origin should always be suspected in an infant who tends to regurgitate his feedings over a long period of time or in an older child who has difficulty swallowing solids. In adolescents and adults the diagnosis is more difficult. Consideration of the patient's history is important. There is usually the fact that the difficulty was first noticed in infancy, with a history of intermittent attacks of acute dysphagia becoming more and more troublesome and more frequent with increasing age. The suppleness and appearance of the esophageal mucosa are characteristic at esophagoscopy. Furthermore, the absence of evidence of inflammatory thickening and the localized valvelike nature of these strictures are typical and should make it possible, with the aid of a careful history, to exclude such diagnoses as stricture from chemical burns, esophagitis with ulceration, and other acquired stenoses.

TREATMENT If there is a coexisting esophagitis the diet should be restricted to liquids and time allowed, with daily lavage and the administration of an antibiotic medication if indicated, for the inflammation and spasm to subside. Gradual instrumental dilatation of the stenosis, often with disruption or avulsion of a web or membrane, can be undertaken and often results in success. In some instances, however, dilatation or disruption of the web or stenosis may appear to be too hazardous because of the danger of perforating the esophagus above, or it may not succeed after trial. In either event, surgical intervention may be required. This is done using a standard thoracotomy incision through the eighth intercostal space on either the right or the left side as preferred. The left side is usually chosen because it is more convenient in the rare case in which a resection has to be carried out. In the average case the esophagus

is opened longitudinally over the stricture and if there is a diaphragm or web present, the obstructing membrane can be excised circumferentially. If the mucosal edges tend to separate at the point of attachment of the web to the wall of the organ, they should be drawn together with a row of 5-0 silk sutures. The esophagotomy incision is then closed longitudinally with a layer of silk sutures in the mucosa and a second layer in the muscularis.

Occasionally the obstructing lesion is more than just a web. In this event the obstruction may be overcome by an esophagoplasty consisting of a longitudinal incision through the constriction and circumferential closure of all layers. If the lesion extends a few centimeters along the esophagus, a resection of the abnormal segment may be necessary followed by an end-to-end anastomosis of the esophagus provided that tension at the suture line can be avoided. If the diseased segment is still longer, an esophagogastric anastomosis must be performed to restore continuity (see Chapter 20).

Malformations Involving the Trachea

Of this group the most frequent is atresia of the esophagus with a tracheo-esophageal fistula of variable size and accompanied or not by abnormalities of one or the other conduit. These cases make up about 70 to 80 per cent of all anomalies of the esophagus. Four anatomical types can be distinguished.

In *Type 1* the proximal segment opens into the trachea and the distal segment forms a blind pouch or cul de-sac (Fig. 105).

In *Type 2* the proximal segment forms a blind pouch of variable length ending anywhere from the neck to the level of the lower portion of the trachea where the distal segment opens separately into the posterior surface of the trachea proximal to its bifurcation (Fig. 106).

Type 3 is a variant of *Type 2*, differing only in the lower segment which opens into the respiratory passage at the carina or into the posterior surface of either bronchus—usually the right (Fig. 106).

Type 4 is characterized by the presence of a double fistula. Both the upper and lower segments open into the posterior surface of the trachea (Fig. 107). An infrequent variant of this type illustrated in Figure 107 comprises an almost complete failure of fusion of the two lateral ridges in the primordial digestive tube, resulting in a fistulous communication between the esophagus and trachea extending practically from the larynx above to the tracheal bifurcation below (Fig. 108).

Of the four types of fistula which may occur, the most common are *Types 2* and *3* in which there is a blind proximal pouch and the fistulous communication is with the distal segment. This arrangement is found in upwards of 85 per cent of the cases of atresia accompanied by a fistula into the respiratory tract.

In these the proximal pouch resembles a diverticulum, averaging 3 to 4 cm. in length. It may extend downward only a short distance but more often it lies in the superior mediastinum though rarely below the level of the tracheal bifurcation. Its diameter averages 12 to 15 mm. It is rather dilated and its walls are relatively thick because of an abundance of smooth muscle fibers. It is often covered by a plexus of dilated veins.



FIGURE 105



FIGURE 106

FIGURE 105 Esophagotracheal malformation (Type 1) The fistulous connection is with the proximal pouch

FIGURE 106 Esophagotracheal malformation (Types 2 and 3) Proximal blind pouch The fistulous connection is with the lower segment at the tracheal carina or slightly above it on the posterior tracheal wall

The distal or inferior segment is elongated and conical as it narrows to the point at which it is attached to the trachea or bronchus. Its fibers beyond the point of attachment tend to blend with the posterior surface of the trachea and sometimes also with the fibers of the proximal segment. This segment is rudimentary, very short, and of a small diameter, rarely more than 5 to 7 mm. It is always much less distended than the proximal pouch. The defect between the two segments varies from 1.5 to 3 cm. Sometimes, however, the distance between them is very great and an anastomosis cannot be made. The optimum distance for a successful anastomosis should not exceed 2 cm.

The fistula is usually found about 5 to 10 mm above the bifurcation on the posterior wall of the trachea (Fig. 109). It is most often a narrow longitudinal slit with a concave, valvelike lower border which looks somewhat like the ureteral orifice in the bladder. It can also be double.

Although this anomaly may exist alone it may also be associated with others in a rather large percentage of the patients. Cardiac abnormalities including interatrial and interventricular septal defects, patent ductus arteriosus, and transposition of the great vessels are the most common. Others include imperforate anus, Meckel's diverticulum, intestinal reduplication or atresia, and abnormalities of the lungs, the thyroid gland, or the skeleton. Horseshoe kidney may be encountered and abnormalities of the genital tract such as bicornuate uterus, double vagina, or the occurrence of a cloaca have been observed.

Finally, there exist even more complex situations. In one instance reported by Collet the trachea ended in the hilus of the right lung while the left bronchus arose from the side of the esophagus (Fig 110)



FIGURE 107

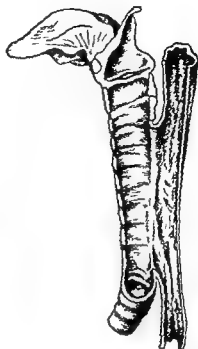


FIGURE 108

FIGURE 107 . Esophagotracheal malformation (Type 4) A double communication one with a proximal the other with a distal segment with no esophageal continuity between

FIGURE 108 Esophagotracheal malformation (modification of Type 4) The esophagus and trachea are united for a considerable distance

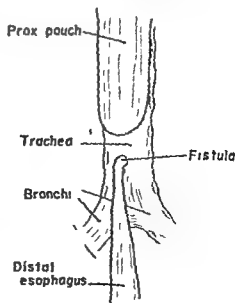


FIGURE 109 Esophagotracheal fistula Type 2 the most commonly encountered arrangement. The lower segment of esophagus opens into the trachea the upper segment ends in a blind pouch

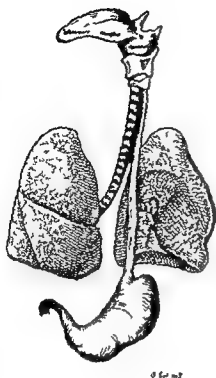


FIGURE 110 . Esophagotracheal malformation. The esophagus is joined with the left main bronchus (complex case of Collet)

Pathogenesis

Little is known about the etiology of these abnormalities. There appears to be a hereditary factor, exemplified by an instance reported in the literature of three infants born of three different mothers but with the same father, and each of the infants had malformations of the esophagus. Many of the anomalies occur in families which tend to have a high incidence of stillbirths.

Clinical Characteristics

In the case of complicated abnormalities, particularly when associated with other malformations, the infant dies soon after birth. With the recent developments in the field of thoracic surgery, however, it may be possible to save a few such infants.

The usual case of congenital atresia with tracheo esophageal fistula is compatible with survival if recognized early and operated upon promptly. The infant, so far as external appearances are concerned, is usually normal. It urinates and passes meconium. On being allowed to nurse, however, as soon as it swallows it suffocates, becomes cyanotic, coughs, and regurgitates the ingested liquid through both the nose and the mouth. Sometimes even before the first nursing the true state of affairs may be suspected by the observation of certain characteristic occurrences. These are the sudden expulsion of very foamy mucous secretions by way of the nose and mouth, uncontrollable paroxysms of coughing, the foamy appearance of the first meconium stools caused by abnormal amounts of air in the intestinal tract, and superabundant salivation. When the infant is allowed to nurse, the same sequence is continued. It takes its feedings avidly but regurgitates the ingested milk immediately in a spasm of suffocation.

On examination of the child no abnormality of the buccal cavity or pharynx may be seen, but the pharynx is partially filled with milk and secretions, and if boiled water is given, cyanosis, cough, temporary cessation of breathing, and vomiting occur. This practice is therefore not to be recommended. Furthermore, the epigastrium is distended and tympanitic because of the large amount of air in the stomach. This combination of physical findings is so characteristic that the diagnosis should be suspected immediately.

Roentgen Examination

The diagnosis is confirmed by the characteristic findings on roentgen examination, but the examination must be made with certain precautions. Fluoroscopic observation should be carried out and films made in the anteroposterior and lateral projections without use of a contrast medium. Associated abnormalities of the lung, notably atelectasis especially of the upper lobes, or pneumonia should be sought for. These may be found in as many as one-half the patients. Cardiac abnormalities may be observed as well.

So far as the esophagus is concerned, it may be possible to detect the air-filled lower segment, but the only conclusive evidence is that obtained by introducing a small amount of a contrast medium into the proximal pouch. This should never be a barium mixture, however, because of the exceedingly irritating nature of this material which, when aspirated into the respiratory passages, leads to severe inflammation and often fatal pneumonitis.

The child should be examined in the recumbent position. With the aid of fluoroscopic control a small rubber catheter is introduced through the pharynx and a small quantity (not over 1 cc.) of Lipiodol is injected. This outlines the proximal pouch, which is seen usually extending to a point between the first and third thoracic vertebrae (Fig. 111). The point of maximum descent of the pouch during inspiration and while the child is crying should be recorded, also its caliber and its depth. Careful search should be made for evidence of the passage of any Lipiodol into the trachea which would indicate a proximal fistula (Type 1 or 4). Films should be taken to establish the length of the proximal pouch and its mobility should be noted. At the completion of these observations, every bit of Lipiodol must be aspirated and a final fluoroscopic check should be made to be sure that this has been accomplished.

Attention is then turned to the abdomen where the presence of air in the stomach and intestines will be observed. The stomach is usually found greatly distended with air because the child breathes directly into it in the usual type of case. Figure 111 shows the characteristic roentgen appearance of an infant with congenital atresia consisting of a proximal pouch and a distal segment fistula causing marked dilatation of the stomach with air.

On the basis of the above examination the exact type of deformity present can be predicted with a high degree of accuracy as shown in Figure 112.

Sometimes however the roentgen examination may be misleading or inconclusive, particularly when the infant regurgitates the Lipiodol and then aspirates it into the trachea even though a proximal fistula is not present. Sometimes also, even in Types 2, 3 and 4, air may not be found in the gastrointestinal

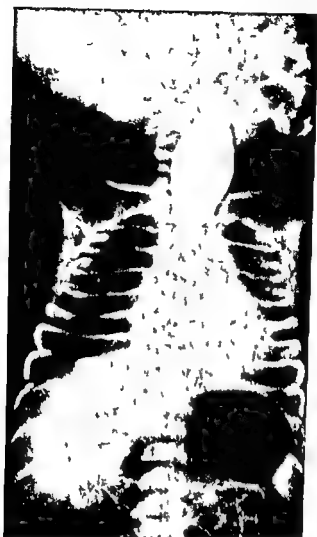


FIGURE 111 Shadow of atresia obtained by ingestion of opaque medium. Note the enormous air filled stomach indicative of a tracheo esophageal communication with the distal segment (Tédesco)

Type	Gastric Air	Lipiodol in Trachea
Malformation of the esophagus without fistula	0	0
Malformation with fistula		
Type 1	0	+
Type 2	+	0
Type 3	+	0
Type 4	+	+

FIGURE 112 Table illustrating the diagnostic aspects of the various types of tracheo esophageal fistula and esophageal atresia

tract because of the extremely small dimensions of the fistulous opening or because the distal segment is occluded even though it connects with the trachea. Thus, in any case in which air is not found in the stomach, the likelihood of finding a situation which can be corrected surgically is greatly reduced except when the opening is unusually small.

Bronchoscopic Exploration

Bronchoscopic exploration to aspirate the secretions and search for the fistula opening has been advocated but with the dangers of this maneuver in a normal infant in mind, it cannot be said that this is not a hazardous procedure in these abnormal and debilitated infants, many of whom are premature. The tracheostomy which might have to be performed because of postbronchoscopic laryngeal edema might provide a certain handicap, although anesthetization for a subsequent surgical procedure can actually be performed through the tube and postoperative clearing of the airway, by frequent aspiration through the tracheostomy tube, is often the best way of preventing aspiration pneumonia.

Esophagoscopy

Though recommended by some clinicians esophagoscopy not only is highly dangerous because of the possibility of perforation and of interference with respiration, but it also does not provide especially useful information.

Except in very unusual circumstances, therefore, both bronchoscopy and esophagoscopy are best not included as a part of the examination of these infants.

Prognosis

Left to themselves these infants die, as Lelong has said, because they swallow into their lungs and inhale into the stomach. Aspiration of liquid into the lungs is the usual cause of asphyxiation, but this may be accentuated by the pressure of a distended proximal pouch against the soft trachea to further occlude the lumen. The signs of pneumonia may be obscured by the other aspects of the case, but pneumonia and asphyxiation are the usual causes of death. Sometimes the child is so weak that it remains somnolent without making any effort to swallow.

Treatment

As soon as the diagnosis is suspected the child should be rushed to a center where proper surgical treatment can be carried out. While being transported it should be kept lying down to favor drainage of secretions from the respiratory passages and it should not be fed orally.

The aim of treatment is threefold:

- 1 To save the life of the infant, since without treatment he will die. For this reason the indications should be liberal and no infant should be denied treatment from the attempt unless he is exceptionally underdeveloped.

- 2 To avoid pulmonary infection, which inevitably results from the inundation of the respiratory tract either with material swallowed into it directly through a fistulous communication or by the aspiration of ingested liquids regurgitated from the proximal pouch.

- 3 To establish esophageal continuity and insure as rapidly as possible adequate nutrition for the child.

CHOICE OF PATIENT FOR OPERATION The most important single factor in the success of the treatment is prompt diagnosis by the physician and immediate consultation with the surgeon. In general as mentioned above the benefits of surgery should not be denied in the majority of instances. There are, however,

certain conditions which should lead to hesitation. If the infant is two or three weeks premature, surgical intervention is of questionable value because of the slight chance of survival. The larger the child, the greater his chance of withstanding the operation. It is wise not to operate upon any infant whose birth weight is under 4 pounds. Infants whose cry is feeble or whose cyanosis persists in spite of aspiration and oxygen therapy offer a bad prognosis. Also, if the temperature continues to climb or stays high, operation should be deferred.

The condition of the lungs is much the most important criterion. If there is bilateral pneumonia, operation is definitely out of the question. It is possible to operate successfully if the upper lobe of one lung only is involved. Likewise, if there is atelectasis, provided it is on the side to be operated upon, it may be possible to proceed. It is difficult, however, to distinguish between atelectasis and pneumonia in some instances in these very small infants.

PREOPERATIVE PREPARATION The preliminary preparation of the patient is important in every instance but especially in a newborn infant whose general condition is not good and particularly when there is a serious degree of pulmonary disease, and with infants who have managed to survive and are seen first several weeks after birth.

Respiratory disorders, whether due to actual infection with lobar pneumonia or aspiration bronchopneumonia or to interference with ventilation resulting in atelectasis, must be treated energetically. Liberal use should be made of the antibiotics and the child should be placed in an oxygen tent with a high concentration of the gas. Furthermore, in order to maintain the respiratory passages free from the accumulation of secretions, continuous suction should be applied to an intranasal catheter (No. 8 Nelaton). Additional assistance in keeping the airway free is to be had by placing the child in slight Trendelenburg position, which permits better ventilation and minimizes swallowing into the trachea.

Finally, suitable use should be made of transfusions of blood or plasma, and infusions of glucose solution or a mixture of electrolytes in physiologic proportions supplemented with ascorbic acid and vitamin K.

This regimen, however, must be carried out with the assistance of frequent laboratory determinations to avoid harmful effects which might result from proceeding blindly. For example, blood should be given if the red cell count is below 4 to 4.5 million and the hemoglobin level is low, however, if there is hemoconcentration, plasma should be administered to improve the protein level. The same is true in the effort to overcome dehydration. Excessive amounts of liquid, especially of saline solution, must be avoided because of the hazards of pulmonary edema in these hypoproteinemic infants who are especially sensitive to overdosage with sodium chloride. The assistance of a skilled pediatrician is indispensable in the management of this aspect of the treatment.

The choice of the correct time to perform the operation must of course depend upon the exigencies of the situation. Ideally, the opportune moment is some time between eighteen and twenty-four hours after the preoperative treatment has been started. At this time usually the temperature shows a tendency to return to normal, the pulse has slowed and the cyanosis has disappeared so that the situation seems to be less alarming. One should not, however, exercise

too much haste, although a delay of over forty-eight hours is usually not wise. The decision is particularly difficult because, while hoping for improvement in the general condition of the patient, one is faced with the mounting seriousness of the pulmonary situation which increases with every moment of delay until the condition can be corrected surgically.

Morphine should never be given preoperatively because of its depressing effect upon respiration, but a preliminary dose of atropine should be administered one-half hour before the start of the anesthesia.

ANESTHESIA In patients whose condition is grave particularly because of the coexistence of pneumonia or pulmonary atelectasis the incision may be made with local anesthesia using procaine hydrochloride. The control of the expansion of the lung is then maintained by administering oxygen through a catheter inserted into the trachea by way of the nose. The airway is made tight by packing the pharynx around the tube. In other instances the total duration of administration of a general anesthetic such as ether can be reduced by using local anesthesia until the thorax is opened and during closure.

In the majority of instances a closed circuit general anesthesia is employed, usually with cyclopropane but sometimes with ether as the agent. With this means there is less operative shock than with local anesthesia and the surgical procedure is technically more easily accomplished. Furthermore, when the pleura is opened either accidentally or on purpose, a means to control the lung is provided. It is striking to observe how rapidly the lung of a newborn infant becomes atelectatic with the chest open. The surgeon should keep this constantly in mind and should request the anesthetist to expand the lung each time it becomes collapsed.

The use of an endotracheal tube is open to criticism because of the possibility of inducing laryngeal and subglottic edema. There is also the objection that the end of the tube might impinge upon the fistula opening. Usually these difficulties can be avoided by using a tight-fitting face mask instead of a tube and a mouth airway with gauze packing around it.

An intravenous infusion of fluid should be kept going throughout the procedure. Through this a transfusion of 100 to 150 cc of blood can be given if indicated.

OPERATIVE TECHNIQUE The surgical approach may be either transpleural or retropleural. The former may be made quickly through an intercostal incision in either the fourth or fifth space. It provides quick access to the region of the esophagus and trachea as well as an excellent exposure but it has the disadvantage that expansion of the lung may be difficult to control and that in case of leakage at the suture line an empyema may result—a complication which may be fatal in these debilitated infants.

The retropleural approach has the advantage that the lung does not collapse unless the pleura is opened accidentally and that leakage, if any, from a faulty anastomosis remains extrapleural. Its disadvantages are that it necessitates the division posteriorly of several ribs to gain adequate exposure and that it takes longer to make and is somewhat more troublesome to close (Fig. 113 2, 3). The right side provides the best approach because it avoids the interference of the aortic arch. Some surgeons, however, have advocated using the left side if the

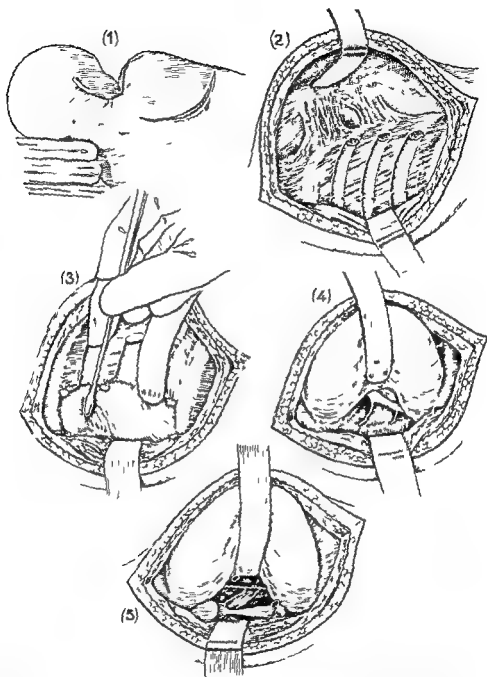


FIGURE 113 : Surgical approach for the correction of esophageal atresia with tracheo-esophageal fistula: 1 Position of the patient and skin incision 2 Exposure of the upper thoracic cage and freeing of ribs Dotted line shows the line of incision of the ribs and interspaces 3 After resection of the posterior portions of the ribs and ligation of the neurovascular bundles lung and pleura are reflected (Note Some surgeons prefer an intercostal incision and transpleural approach avoiding the division of ribs) 4 Exposure of the azygos vein indicating that reflection of the lung and pleura is almost completed 5 Wide exposure of the proximal pouch and tracheo-esophageal communication obtained (After R. Gross)

lung on that side happens to be affected either with pneumonia or atelectasis so as to spare the uninvolved lung to maintain better oxygenation during the procedure

The infant is placed on his left side with the right side arched slightly by placing a pillow or a folded blanket beneath. The right arm is held in abduction to swing the scapula away from the operative field. If the transpleural approach is employed, an incision is made in the fourth interspace. If the extrapleural method is chosen, a 1 cm segment of the posterior portion of each of the second, third, fourth, and fifth ribs is excised; the intercostal muscle bundles are divided, and the vessels and nerves are ligated and divided. The pleura is dissected away from the chest wall, uncovering first the sympathetic chain and then the azygos vein which is divided between ligatures.

After a few moments devoted to the examination of the characteristics of the anomaly, the proximal pouch is identified and dissected free as far upward as the level of the first rib. This can be done without fear of injuring its blood supply, which is ample. The lower segment is then identified. This may be somewhat more difficult than the preceding step because of the small caliber and collapsed state of this portion of the esophagus. In the usual case it is this portion which is attached to the trachea or one of the bronchi. In separating the two organs at this point, great care must be exerted not to injure the thinned-out esophageal wall, every bit of which must be preserved. The fistulous connection is severed, cutting actually in the wall of the trachea with a sharp knife so as to preserve as much length of the distal esophagus as possible.

The opening is then closed with several sutures of fine silk, preferably the type used for arterial anastomosis with the thread swaged onto the needle (atraumatic). This closure should be covered with an outer layer of areolar tissue or a flap of pleura if the transpleural approach is used.

Once the tracheal opening is closed, the decision must be made as to the possibility of performing an end-to-end anastomosis of the two segments of the esophagus. In recent years it has been shown that unless there is partial occlusion of the distal segment or unless the proximal pouch is unusually short, an anastomosis can be accomplished in the majority of the patients even when it may appear at first to be impossible. To bring this about an extensive mobilization of the lower segment is carried out, but in doing this caution must be observed to preserve the integrity of the aortic arterial branches which carry much of the blood supply. These vessels slope downward, however, and as the segment is gradually freed from its mediastinal and diaphragmatic attachments they tend to swing up, making it possible to pull the esophagus up an appreciable distance without injuring them. The arteries which supply the lowermost portion of the segment (branches of the inferior phrenic and left gastric arteries chiefly) must not be injured. They can usually be avoided.

If, however, after every degree of mobilization has been secured and there is still no likelihood of bringing the ends together without tension on the anastomosis, the attempt must be abandoned. In this event the end of the distal segment is inverted with Lembert sutures to avoid retrograde leakage from the stomach. The chest is then closed and the proximal pouch is brought out through an incision in the left side of the neck where it is fastened with sutures.

An immediate opening is made into the end of the pouch to prevent subsequent aspiration into the respiratory tract. At the same operation or, if the patient's condition is not good, on a subsequent day a gastrostomy is performed. These maneuvers comprise the first stage of a two stage restoration of continuity, a procedure rarely necessary today. The second stage is then accomplished after the child has reached the second or third year by one of several methods of esophageal reconstruction to be described.

Primary Anastomosis (One Stage Procedure)

Once it has been established that the ends can be brought together without undue tension, the anastomosis is begun. As a first step the end of the proximal pouch must be opened without sacrifice of length. The technical difficulties to be overcome are chiefly the problem provided by the extreme thinness of the wall of the distal segment and the often great disparity in size between the diameters of the two ends. In no other procedure having to do with the esophagus is it more important to avoid traumatism of the tissues and interference with their blood supply. The ends of the segments should be subjected to a minimum of handling, they should not be grasped with forceps or any instrument which might produce tearing, and the sutures must be placed accurately and tied not too tightly. Atraumatic arterial sutures of 5-0 or 6-0 silk attached to small curved needles should be employed. Several variations of technique have been advised. The method suggested by Haight has great merit. It consists of two layers. The deep or inner layer is made by suturing the full thickness of the edge of the distal segment to the mucous membrane layer of the proximal segment, using interrupted silk sutures. For the outer layer the edge of the muscular coat of the proximal segment is sutured to the outer surface of the muscularis distal to the first layer (Fig. 114).

An ingenious modification of technique suggested by Gross has the advantage that it makes possible an anastomosis of larger diameter with less tendency to subsequent stenosis and also conserves the length of the segments. It consists of making an oblique opening at each end as seen in Figure 115. The sutures are then applied in the usual fashion. Gross uses a method of anastomosis first developed by Ladd, with an inner layer of stay sutures placed far apart and an outer layer of ordinary sutures placed close together (Fig. 115).

Once the anastomosis has been completed, the tube lying in the proximal segment must be withdrawn. It is unwise to insert a tube through the anastomosis, even though this provides a capital way of feeding the patient, because of the risk of damage to the suture line resulting from pressure necrosis of the delicate tissues. There is also the danger of increasing the risk of respiratory infection because of the presence of the tube in the pharynx.

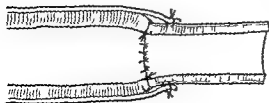


FIGURE 114 Haight technique for anastomosis

tion must be parenteral, using the intravenous route. A balanced solution of electrolytes containing dextrose is used. Blood should be administered as indicated. The protein level of the blood must be examined as well as the plasma electrolyte determinations. Plasma or human albumin may be given if the protein level is low. If the hemoglobin level is low, blood is administered. Great care must be exerted to avoid the development of tissue edema. Pulmonary edema may prove fatal in these feeble subjects.

If after forty-eight hours difficulty is experienced in maintaining the proper hydration, protein levels, and so forth, a small rubber tube should be inserted under local anesthesia into the stomach. Through this a nourishing liquid formula may be administered, starting eight hours after the gastrostomy was established. Small amounts are given every two hours at first to avoid overdistention and subsequent vomiting which might compromise the anastomotic suture line.

Approximately ten days after the operation, if everything is going well, 1 or 2 cc of Lipiodol may be given by mouth and the esophagus observed under the fluoroscope. If no fistulous tract is seen and if the lumen at the anastomosis is open, oral feedings may be started, beginning with a spoonful of sterile water each hour. Sterilized milk may then be tried and within a few days the amount of formula given by mouth can then be increased to the point where all nutrition is supplied in this manner. The gastrostomy tube is withdrawn as soon as it is obvious that it is no longer needed.

COMPLICATIONS Complications are chiefly pulmonary or pleural. Atelectasis from bronchial obstruction by retained secretions may be serious if an entire lung is involved or if two or more lobes are affected bilaterally. As soon as the secretions are eliminated, re-expansion usually takes place promptly. Pneumonia is a frequent cause of postoperative death. Suitable antibiotic medication should be administered.

If the pleura is opened either intentionally or accidentally, at operation, persistent pneumothorax can be prevented by thorough expansion of the lung on the part of the anesthetist as the chest is being closed. If a pneumothorax should be discovered during the postoperative period, prompt aspiration relieves the situation. Pleural effusion may develop. This is more often seen when the transpleural approach has been employed than with the extrapleural approach. The fluid should be removed by thoracentesis as soon as it is recognized.

Roentgen films of the chest should be made whenever respiratory difficulty becomes a problem, using a portable apparatus if the child is very ill, as he is likely to be.

Leakage at the line of anastomosis may develop either because of a technical error, unavoidable tension on the suture line, or failure to heal in a debilitated patient with nutritional deficiencies. Avoidance of vomiting is obviously of paramount importance. If small, these fistulae usually heal of themselves but subsequent stenosis due to cicatricial contraction may occur and bougienage may become necessary. In an occasional instance a second operation may be required to correct the difficulty.

RESULTS Although the percentage of failures with primary anastomosis was formerly high, improvements in surgical technique, anesthesia, and the general care of the patient have brought about a striking reduction in the post

operative mortality which under ideal conditions now should not exceed 10 per cent. The operation, however, as mentioned in detail above should not be employed unless the condition of the patient is reasonably favorable to begin with.

Two Stage Procedure

When confronted by an anatomical situation in which it is impossible to perform a primary esophageal anastomosis, it is never wise to attempt a reconstruction using stomach or intestine as a primary procedure. The infant is rarely strong enough to withstand the shock of so extensive an operation at that time. Furthermore, the presence of stomach or intestine in the relatively small chest of the newborn interferes too greatly with the respiratory function because of the space occupied by the transplanted viscus. This difficulty is accentuated particularly if the stomach is used, because of the enlargement produced by the large volume of liquid nourishment which makes up the normal dietary requirements of the patient. A stomach full of milk lying in the chest of such a small infant may reduce the pulmonary volume to such an extent that breathing is impaired.

It is best, therefore, in such a case to resort to a two stage operation. As mentioned above, the proximal pouch should be exteriorized in the neck immediately. The gastrostomy for feeding may be postponed until the patient shows evidence of recovery from the operation. *This comprises the first stage of the procedure.*

The second stage, at which some method for restoration of continuity is carried out, should be delayed until the child is at least two years of age. Formerly the method usually employed was the construction of an antethoracic skin tube. Relatively few notable successes were ever obtained with this method. The technique is prolonged, involving several stages for its completion, and fistula or stricture formation, particularly at the point where the lower end of the skin tunnel is attached to the stomach, are frequent occurrences. In addition this tube once established is unsatisfactory, both esthetically and functionally. The mole track appearance of the front of the chest is unsightly and embarrassing especially in girls, and the annoyance of having to wipe the ingested food downward with a sweep of the hand each time something is swallowed is likewise embarrassing. The emotional and psychological complications of a situation such as this can readily be imagined. This method therefore is highly undesirable and should not be attempted.

The ideal procedure is the substitution of either the stomach or a segment of intestine in place of the absent esophagus. To avoid the unsightly appearance of a subcutaneous transplant, the transplanted viscus should be brought up through the mediastinum or in some instances through one of the pleural cavities. Thus from both the cosmetic and the functional points of view the necessities of the patient's condition are better met by this method. The actual technique of the procedure is described in Chapter 20.

RESULTS The stomach has been employed in a series of patients with gratifying results in spite of the theoretical objections to its use. Some of these patients have been observed over a long period of time, one actually ten years



FIGURE 116

FIGURE 117

FIGURE 116 Photograph of patient W C, operated upon by the two stage technique using the stomach for esophageal replacement. Note 1, Upper cervicothoracic incision for the esophagogastric anastomosis 2, closure of gastrostomy 3, anterior end of the intercostal thoracotomy incision. Age 21 months

FIGURE 117 Recent photograph of patient, W C, at the age of 12 years showing normal development

(Figs 116 and 117) As stated earlier, the children who have been fed all their lives by gastrostomy may have to learn to eat, and the amount of food consumed at first may not be sufficient. This difficulty may be obviated by encouraging them to chew and swallow crackers or other suitable foods during the interval between the stages of the operation. Ultimately after the initial period of readjustment the children who have had this type of operation have apparently developed normally, both physically and mentally. No cases of esophagitis have been observed and there appears to have been no difficulty with regurgitation. No case of anastomotic stricture has been observed in the series studied. Likewise the patients do not experience any subjective awareness of the presence of the stomach within the chest.

There are some advantages, however, to the use of either the jejunum or a segment of colon to supplant the esophagus in these children. The employment of the jejunum presents greater technical difficulties than the use of the colon. The advantages are that the vagus nerves do not have to be interrupted and the stomach is left in its normal location. (For the technique see Chapter 20.)

The use of either jejunum or colon, however, is attended by a higher incidence of complications than when the stomach is employed. Anastomotic fistula formation is frequently encountered and sloughing of the end of the trans-

planted segment of bowel may occur With the colon this has been reported as a late complication occurring several months after the operation and resulting from unexplainable failure of the blood supply of a colon transplant In the absence of complications the functional behavior of a colon transplant is satisfactory With the jejunum, however, there may be nutritional deficiencies particularly if the transplanted loop is not connected with the stomach The food then enters the small bowel directly, by passing the stomach, and the attendant discomforts and complicating diarrhea which may result are troublesome (See Chapter 20)

Congenital Short Esophagus with Thoracic Stomach (Brachyesophagus)

The true significance of this condition has been recognized only in recent years Too frequently still the existence of a short esophagus is considered to be an acquired condition resulting from ulceration and cicatrization whereas in fact with few exceptions the reverse is the case This condition to which Lelong has applied the term *brachyesophagus*, presents a constant and characteristic anatomical pattern The esophagus is abnormally short, reaching in the majority of instances as far as the lower mediastinum several centimeters above the diaphragm Sometimes however, it may be much shorter ending at or just below the aortic arch At its lower end it merges with a cylindrical segment of stomach often no larger in diameter than the esophagus itself which extends below the diaphragm to join the body of the stomach in a manner which bears no resemblance to the usual appearance of the cardia Rather the cardia in this condition is represented merely by the point of transition usually abrupt

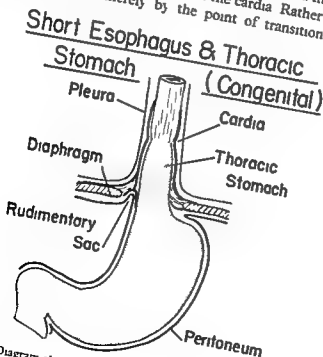


FIGURE 118 Diagram showing the anatomy of congenital brachyesophagus

between the squamous type of mucosa of the esophagus and that characteristic of the stomach. When viewed externally, as at a surgical operation or autopsy, the difference between the tubular segment of the stomach and the esophagus itself is always readily recognized, although the line of demarcation may not correspond exactly with the point of mucosal transition. Sometimes, in fact, the gastric mucous membrane appears to extend several centimeters into the esophagus itself.

The relation of the diaphragm to the stomach is usually normal, although in some instances a small pouch of peritoneum may be identified extending up a short distance in front of the thoracic segment of stomach. The condition, however, should not be regarded as a hernia. It is a developmental anomaly involving the esophagus and the proximal portion of the stomach (Fig. 118).

Etiology

Because of its congenital nature, the origin of this abnormality is obscure. It is often familial. Other members of the same family may have the same or similar abnormalities of development of the diaphragm or esophagus. It is sometimes seen in persons with Marfan's syndrome. It may be accompanied by pyloric stenosis, inversion of the stomach, malrotation of the intestines, shortness of the colon, etc. It is more common in boys than in girls.

Pathogenesis

The development of this condition has something to do with abnormalities of the interdependent evolution in the early embryo of the diaphragm, the esophagus, and the spine. In order to understand this, a brief description of the development of the diaphragm may be helpful. The development of the diaphragm is a complicated evolution involving the growth and final fusion of three elements: (1) The first is the *transverse* (or *pericardioperitoneal*) *septum* which appears very early and hangs directly from the liver bud. This is the primordial diaphragm. It lies in a ventral position. (2) The second is the *pleuroperitoneal septum* arising in the primitive dorsal mesentery. This is, therefore, dorsal in position and appears later than the former. (3) The third is a layer of *mesenchymatous* origin which develops from the sides of the trunk and by connecting the other two septa and, uniting in the midline, brings about the final separation between the two thoracic cavities and the abdomen.

During embryonic life, important variations in the *metameric* position of the diaphragmatic muscle are observed. By the middle of the second month, profound changes supervene when cartilaginous segmentations of the spinal membrane begin to differentiate to form the origins of the future vertebrae. The first of these to appear are in the dorsolumbar region.

At the same time the esophagus, which at the beginning was represented, as we have seen above, by a rather short canal between the pharyngeal bulge and the stomach protrusion of the primordial digestive tract, grows rapidly to keep pace with the development of the neck and the thorax. Furthermore, the esophageal musculature does not appear until the end of the second month. During the second month there appears to be a critical phase in the development

of the diaphragmatic segment of the esophagus occurring at a zone corresponding to the point where it encounters the transverse development of the diaphragm. Its early fixation at this point is interdependent upon both the caudal migration of the diaphragmatic muscle and the vertebral segmentation of the spine. The latter is subject to numerical variations particularly in the thoracic segment, although this is not generally recognized.

In summary the esophagus is obliged in its development to adapt itself to the diaphragm and the spine. The former intervenes with its variations and the latter because of its role in the metamerization and numerical variations of the dorsal segment. In other words, a brachyesophagus results from a dwarfism of the primitive gut which is essentially conditioned by the spine and the diaphragm. The superior pole of the stomach which remains drawn up in order to meet the lower esophagus, of necessity becomes fastened in the diaphragmatic canal or hiatus.

Clinical Characteristics

Brachyesophagus frequently does not cause symptoms and may be discovered only accidentally in later life.

BRACHYESOPHAGUS IN CHILDREN The usual manifestations are those of either partial obstruction or anemia, or both. The condition is usually recognized during the first few days after birth but it may not cause symptoms until after a more or less prolonged latent period. The earliest manifestation is vomiting, appearing usually during the first nursings but sometimes not until about the second month or even as late as the sixth month when the transition to solid food is begun. The periodicity of the vomiting spells is often irregular. Sometimes they occur every time the child is fed, and in such a case death usually ensues from inanition. Sometimes several days may pass without any difficulty only to be followed by repeated attacks. At other times only certain meals are lost. The vomiting bears no relation to the type of feeding or formula given. It is less troublesome with thickened mixtures but does not always disappear as a result of altering the physical characteristics of the food. The vomiting may be violent or there may be only slimy regurgitations. Often there is accompanying pain. The appetite remains unimpaired and the child will usually take the breast or nursing bottle again after the vomiting is over. On the other hand as with certain other malformations of the esophagus, vomiting may not be an outstanding symptom. The diagnosis often must be made on other evidence than vomiting alone.

Another characteristic symptom is the regurgitation of blood clots or of secretions containing streaks of blood. Sometimes the vomitus is chocolate colored or contains material which looks like coffee-grounds. The quantity of blood vomited is never large. These bouts of hematemesis may be isolated occurrences or they may recur with great regularity.

Melena is sometimes observed either grossly or on chemical test of the stools. Blood is often found in the stools even if the child appears otherwise normal.

The cause of the bleeding is not always obvious. It may be due to repeated traumatism of the hyperemic mucous membrane of the thoracic segment of the



FIGURE 120 Roentgen film of brachyoesophagus of an infant with the cardia closed showing the folds of the gastric segment below it (Jutras)

in color between the pale esophageal mucosa and the reddish mucosa of the stomach below. When esophagitis is pronounced in the portion of esophagus above the stricture, the usual appearance as described elsewhere is recognized. After aspiration, a zone of narrowing can often be recognized and if the esophagoscope can be passed through the cardia, the tubular segment of stomach below it can be seen. In some instances there is no narrowing of the esophagus and no esophagitis, but a severe degree of gastritis may be found in the thoracic segment of stomach.

Differential Diagnosis

Differential diagnosis must be made between brachyoesophagus and an esophageal hiatus hernia. The two conditions are often confused in the minds of the clinician and even of the roentgenologist, although they can usually be distinguished by roentgen examination. In true congenital short esophagus with thoracic stomach the gastric portion is more cylindrical and the esophagus less redundant than in a hiatus hernia in which the esophagus is never short.

The similarity between these conditions is superficial. Nosologically speaking, there is a striking distinction between them. Brachyoesophagus is due to an agenesis of the subtracheal segment of the esophagus and the stomach is in the thoracic cavity because of the shortness of the esophagus. Contrariwise in the hernia cases the esophagus is normal, but there is an abnormality of the diaphragm. Although the stomach may be the cause of symptoms in both types of case, there remains the fact that the prognoses as between brachyoesophagus and hiatus hernia are vastly different. One fact is well established. Brachyoesophagus (short esophagus with thoracic stomach) is a malformation which possesses an anatomical aspect and clinical characteristics which can be clearly differentiated. In practice, with a nursing who vomits repeatedly, this condition should always be kept in mind.

Prognosis

This condition is an abnormality which is often compatible with life and, as already mentioned, may exist without being recognized. The first months are most to be feared but if the infant survives, he usually adjusts to the situation and grows to adulthood without serious setbacks so far as the esophagus is concerned. As the child begins to sit and especially to stand and walk, the vomiting attacks become less and less frequent and the amounts regurgitated smaller.

The evolution of the case may, however, be very capricious. Sometimes the symptoms stop abruptly, the food is no longer vomited and the child begins to put on weight rapidly. However, when these patients are examined in later years the same characteristic malformation is found. In other words, the cure is functional, not anatomical, and this is of course exactly what would be expected.

On the other hand, if the case becomes complicated by the development of esophagitis with ulceration, bleeding, dysphagia, and pain the outlook is very different. When the inflammation is pronounced the child may die of malnutrition, dehydration, and anemia. Because the amount vomited is small these children do not succumb to the usual complications of large congenital hiatus hernias, namely suffocation. Also they do not experience incarceration, obstruction and perforation of the stomach. The unfavorable course of the case complicated by esophagitis can only be overcome by resort to surgical resection.

In the adult, short esophagus may, likewise be complicated by esophagitis, ulcer, stricture, and hemorrhage. There appears to be also a higher incidence of esophageal carcinoma in patients with this abnormality. Resano reports an incidence of nine cases in fifty patients with brachyoesophagus (18 per cent). In a personal experience, 15 per cent of patients with carcinoma of the cardia had a truly short esophagus with a thoracic segment of stomach, an incidence which is certainly greater than the occurrence of this anomaly in the population at large. These observations suggest a much greater incidence of carcinoma in these patients than is generally recognized.

Treatment

With infants it may be helpful to try the effect of posture in preventing regurgitation. The child should be held upright while nursing and for two hours thereafter. It may be helpful also to alter the consistency of the feedings to make them thicker by adding milk concentrates. At about the sixth month, boiled milk and semisolids may be added to the diet. The anemia is combated by the administration of iron or, if severe, by transfusions of blood as indicated to bring the hemoglobin level to normal. Liver extract may be helpful chiefly because of its vitamin content.

If esophagitis develops, alkalis, bismuth or demulcents like Gelusil may be given. Tincture of belladonna or related antispasmodics may relieve the reflex esophagospasm. If the dysphagia becomes severe enough to interfere with nutrition, gentle dilatation may be tried but it should be remembered that in infancy this is a dangerous undertaking. If medical measures fail surgical resection may become necessary.

In adults the usual symptomatic treatment with Gelusil, atropine, and bland soft or liquid diet should be advised. If the stenosis becomes severe because of edema and swelling, gentle dilatation with a bougie passed under direct vision, or with the aid of a thread previously swallowed, is often very beneficial. This must be done, however, without force and tearing of the tissues must be avoided. Bougienage, however, is unwise when a true cicatricial stenosis resulting from ulceration has become established. The treatment then becomes surgical.

Surgical treatment is indicated when there is a cicatricial stenosis, in patients with intractable pain unrelieved by medical means, and when there is severe repeated bleeding. Vagotomy is of no value in spite of the theoretical advantages. Resection has given excellent results in these patients, with a minimum of complications and sequelae. For the details reference should be made to Chapter 13 on Esophagitis.

Congenital Cysts and Reduplication

As a manifestation of abnormalities of the pinching off of the esophagus from the trachea in the early embryo, there may develop in the wall of the esophagus or alongside it a reduplication or accessory tube. The anatomical relations of the latter peculiarities vary. They may communicate with the lumen of the esophagus or they may be occluded at one or both ends. In some instances the only manifestation is the occurrence of a cyst lying in the wall of the esophagus which on removal is found to have a lining characteristic of that of the esophagus or stomach, or of mixtures of both. Symptoms may be nonexistent. Occasional bouts of dysphagia may be experienced, especially when a large bolus is swallowed. Pieces of meat may lodge in this way. In some instances, however, pain, fever, and dysphagia may be experienced, the result of a superimposed infection. The cyst or reduplication under these circumstances behaves like an abscess, and surgical intervention to cure the condition becomes difficult and hazardous. The prevention of this complication is an important indication for surgical removal of the anomaly.

The diagnosis in case there is a connection with the lumen may be suggested at roentgen examination by the escape of barium mixture into the adjoining tract, giving the appearance of a fistula. More often the appearance is that of a mediastinal tumor lying beside or pressing upon the esophagus. With intramural cysts the appearance is that characteristic of a benign tumor of the wall of the esophagus where the barium meal cascades over the mass, fanning out in a typical fashion as exemplified in Figure 121. There is usually no evidence of obstruction.

Surgical removal should be advised to establish the diagnosis and to prevent the possibility of infection. At operation the tubular reduplications can be excised and any openings into the lumen of the esophagus closed. Intramural cysts when uninfected can be excised locally, usually without making an opening into the lumen. When the defect in the muscular layer is closed, the esophagus is restored to an essentially normal state. If the cyst has become infected,



FIGURE 121 Roentgen film of a congenital reduplication cyst in the lower esophagus (man age 54)

usually with surrounding mediastinitis it is necessary to perform a resection, dividing the esophagus through normal tissue above the lesion. Restoration of continuity is effected by esophagogastric anastomosis.

CHAPTER 7

Alterations in Position, Changes in Contour, and Compression

Deviation of Position

The normal course of the esophagus and its relations to the other structures of the neck, the thorax, and the diaphragm have already been described. Although it is relatively fixed in its cervical portion, it is very mobile in the mediastinum where, with the exception of fibrous bands which extend to the trachea and spine in the region of the aortic arch, it is surrounded by an abundance of loose connective tissue. This movability is necessary for the mechanism of deglutition and makes it possible for the esophagus to adapt itself to the configuration of the heart and aorta. It explains also the occurrence of striking degrees of deviation secondary to pathological changes in any of the structures with which it is in relation.

Congenital deviations of the esophagus secondary to malformations of the thoracic spine or any of the mediastinal organs exist, but acquired deviations are more frequent. The shift in position may result either from pressure against it or from traction upon it, or sometimes from both.

In the neck, unilateral enlargements of the thyroid gland may cause a displacement of the esophagus as well as of the trachea. Occasionally branchiogenic cysts or neoplastic involvement of the lymph nodes may be responsible. In rare instances arthritic proliferations of the cervical spine may cause irregularities in the contour of the esophagus (Fig. 122).

In the mediastinum, several causes of alteration in the course of the esophagus may be seen. Deviation or angulation may result from the pressure of tumors, enlargements of periesophageal lymph nodes either from metastatic neoplastic involvement (lymphoma or granulomas due to sarcoid or tuberculosis), or occasionally tumors of the lung presenting on the mediastinal surface, particularly the right (Figs. 123 and 124).



FIGURE 122 Deviations of the cervical esophagus by pressure from arthritic prominences of the spine (Parks)



FIGURE 123 Compression of the midesophagus by a mass of enlarged mediastinal lymph nodes



FIGURE 124 Compression of the upper thoracic esophagus by a mediastinal tumor

Cardiac or vascular enlargements, particularly the left auricular dilatation of mitral stenosis, are a frequent cause of deviation of the esophagus (Fig 125). On its anterior surface the lower esophagus is in relation with the heart and great vessels at the base of the heart. We have seen that the aortic arch normally produces an indentation on the left anterolateral aspect of the esophagus. Enlargements of the aorta in this region, particularly aneurysmal dilatation, may produce a striking angulation or deviation in the course of the esophagus (Fig 126).

With the exception of pressure by large pulmonary tumors presenting on the mediastinal surface the effect of pathological conditions of the lung upon the course of the esophagus is to pull it toward the affected side. This may be the result of the contraction of adherent scar tissue as in the case of fibrotic apical pulmonary tuberculosis, or the deviation may be merely a part of the shift of the mediastinum towards the side of a lung collapsed because of a closed bronchus. An example of shifting of the esophagus from its normal course because of massive atelectasis of the lung is shown in Figure 127.

So far as the spine is concerned minor curvatures have little effect upon the position of the esophagus. Although the aorta usually follows the curvature of the vertebral column rather closely, the esophagus tends to assume a more nearly straight line in the direction of the stomach. Severe degrees of kyphoscoliosis however, may result in a certain amount of esophageal deviation. This is exemplified in Figure 128 where in a patient with marked kyphosis the esophagus takes an unusual backward curve, though not as pronounced as that of the spine. In Pott's disease of the spine the esophagus may become angulated by the pressure of a cold abscess which behaves like a mediastinal tumor in this respect (Fig 129). Severe angulations do not occur, however, unless the esoph



FIGURE 125 . Deviation of the lower thoracic esophagus caused by enlargement of the left atrium in a case of mitral stenosis

agus becomes attached to a portion of the spine by inflammatory adhesions. In the cervical region the esophagus may be pushed out of its normal course by dislocations of the vertebrae—the result of trauma.

Clinical Characteristics

Ordinarily these abnormalities of position, whether produced by pressure or contracture, cause few if any symptoms. In many instances, however, the patient experiences at the time of deglutition a constant or intermittent, often painful sensation referred to the base of the neck or to the upper part of the chest. Sometimes regurgitation or even vomiting may occur after eating. Rarely there may be a severe burning pain felt in the substernal region. It should be remembered, however, that frequently these symptoms are in part the result of the underlying disease, particularly in patients with systemic or toxic manifestations of advanced tuberculosis. In the tuberculous patient also, moderate dysphagia may be experienced from deviation caused by collapse therapy.



FIGURE 126 Compression of the esophagus by an enlarged aortic arch

Roentgen Examination

Quite obviously the diagnosis of these abnormalities of position of the esophagus rests almost entirely upon the roentgen examination. In tuberculosis they will be found mostly in the upper mediastinal segment except for those in the midportion caused by traction from adherent diseased subcarinal or retro bronchial lymph nodes. In general, the deviation follows that of the trachea. The most diverse patterns may be observed. Sometimes the trachea and esophagus cross each other like the letter X. Sometimes sharp angulations may be seen, often in the direction of the sternoclavicular joint. In the hilar region of the lung marked deviations may be seen, but more on the right side than on the left because of the interposition on the left of the aortic arch. In all instances these abnormalities of the esophagus tend to appear accentuated when the patient is requested to cough.



FIGURE 127 Deviation of the thoracic esophagus caused by mediastinal shifting due to atelectasis of the left lung



FIGURE 128 Deviation of the esophagus due to kyphoscoliosis



FIGURE 126 Compression of the esophagus by an enlarged aortic arch

Roentgen Examination

Quite obviously the diagnosis of these abnormalities of position of the esophagus rests almost entirely upon the roentgen examination. In tuberculosis they will be found mostly in the upper mediastinal segment except for those in the midportion caused by traction from adherent diseased subcarinal or retro bronchial lymph nodes. In general, the deviation follows that of the trachea. The most diverse patterns may be observed. Sometimes the trachea and esophagus cross each other like the letter X. Sometimes sharp angulations may be seen, often in the direction of the sternoclavicular joint. In the hilar region of the lung marked deviations may be seen, but more on the right side than on the left because of the interposition on the left of the aortic arch. In all instances these abnormalities of the esophagus tend to appear accentuated when the patient is requested to cough.



FIGURE 127 Deviation of the thoracic esophagus caused by mediastinal shifting due to atelectasis of the left lung

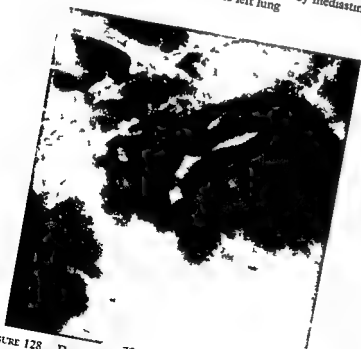


FIGURE 128 Deviation of the esophagus due to kyphoscoliosis



FIGURE 129 Compression of the esophagus by a cold abscess in a patient with Pott's disease of the spine (right anterior oblique view)

Treatment

In general, because they are not usually productive of symptoms, deviations of the esophagus from its normal position do not require treatment of themselves. If they are caused by a condition which can be corrected surgically, as for example by the removal of a mediastinal tumor or a goiter which may be causing pressure or by extirpation of a lung collapsed because of the presence of a carcinoma of the main bronchus, the esophagus is usually restored to its normal relations by the operation.

Localized Compression

Special consideration should be given to certain conditions which cause localized compression of the esophagus.

In the *cervical segment* the esophagus may be compressed by the thyroid gland, primary tumors like branchiogenic cysts or carcinoma, tumors of the larynx or trachea, masses of enlarged lymph nodes, a retro esophageal abscess,

either acute or chronic and in exceptional cases by a large diverticulum filled with food

Thyroid masses are the most frequent. In the ordinary goiter esophageal compression is unusual, but sometimes one lobe may enlarge posteriorly and actually press in between the esophagus and trachea to produce marked compression and angulation (Fig 130). Sometimes both thyroid lobes will enlarge posteriorly to surround the esophagus and constrict it. In the rare case in which a goiter descends into the posterior mediastinum, deviation of the upper thoracic esophagus is a common finding (Fig 131).

In carcinoma of the thyroid the trachea is usually involved first and the

FIGURE 130 : Deviation of the cervical esophagus produced by a unilateral goiter

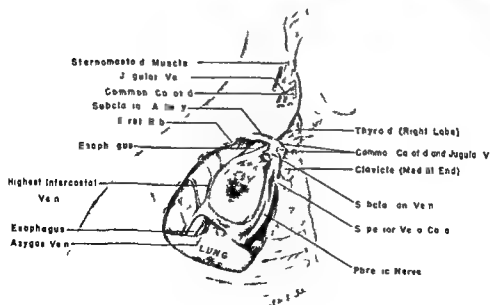


FIGURE 131 Drawing made to show the relations of a posteriorly descending intrathoracic goiter to the superior mediastinal segment of the esophagus



FIGURE 132 Deviation and compression of the esophagus produced by enlarged mediastinal lymph node



FIGURE 133 Compression of the esophagus by a mass of lymph nodes invaded by metastases from a carcinoma of the lung. Note shadows produced by metallic clips placed at the time of exploration to aid in the centering of rays for irradiation treatment

esophagus is thereby spared. In fact, the esophagus is rarely invaded by the primary tumor. Lateral lymph nodes, the seat of metastases, will of course press upon or invade the organ. The manifestations of tracheal obstruction are usually noticed first, but finally there may be a degree of dysphagia as well. Esophagoscopy and particularly esophagoscopy biopsy are contraindicated.

In patients with a neglected large diverticulum of the pharyngo-esophageal junction, compression and obstruction of the esophagus when the pouch becomes filled with food, may lead to severe degrees of malnutrition because the cervical esophagus remains obstructed so much of the time (see Chapter 11).

In the *thorax* the esophagus is so mobile that it is usually merely pushed aside and rarely becomes compressed. If it should be adherent, however, the effect of pressure is always to constrict or obstruct it. Mediastinal tumors and masses of diseased lymph nodes have been mentioned as causes of deviation. If there are adhesions or if there is malignant invasion, obstruction of the esophagus will ensue (Fig. 132). In rare cases dysphagia from compression by involved lymph nodes may be the first sign of a carcinoma of the lung (Fig. 133). Nodes involved by metastases from breast or other carcinomas may produce the same result.

Treatment

Treatment is directed toward removing the cause of compression. When dealing with an inoperable malignant tumor or with mediastinal lymph nodes invaded by metastases the problem is exceptionally difficult. If the constricting mass of tumor happens to be radiosensitive, valuable palliation may follow the employment of radiation treatment. In the majority of instances, however, the relief is only temporary or the treatment may fail entirely to give relief. Unfortunately, surgery has little to offer. Sometimes it is possible, often only with the aid of dilatation, to insert an intraluminal prosthesis such as the Souttar or Markel tube. These if they do not pass down the esophagus occasionally bring about striking relief of the dysphagia. In patients whose general condition is exceptionally good it may be worth while to attempt a by-passing procedure by mobilizing the stomach to effect an esophagogastric anastomosis proximal to the growth or by using a loop of jejunum. In a few instances this type of procedure followed by irradiation of the obstructing growth has produced enough comfort and prolongation of life to make its employment worth while for the patient. If all else fails, a gastrostomy may have to be performed to relieve the intolerable thirst produced by a total obstruction of the esophagus.

The most frequent source of trouble in the thoracic segment is the *aorta*, either because of a congenital anomaly or because of acquired disease like aneurysm.

Compression of the Esophagus by Congenital Vascular Malformations (Dysphagia Lusoria)

The term *dysphagia lusoria* was invented by Bayford in 1789 and applied in a case of progressive dysphagia beginning in infancy in which he discovered at autopsy an abnormal right subclavian artery which arose from the descending

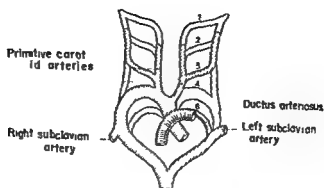


FIGURE 138 Diagram of the embryonic origin of a right aortic arch (After Krause)

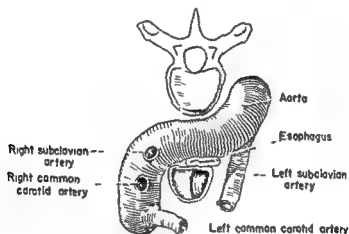


FIGURE 139 Relations of a right aortic arch passing behind the esophagus to descend on the left

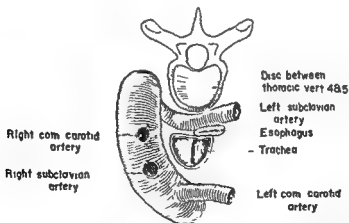


FIGURE 140 Relations of a right aortic arch which descends on the right, shows compression of the esophagus by the left subclavian artery which passes behind it

bronchus lateral to the trachea before curving to the left behind the esophagus to become the descending portion. In some instances of this sort the aorta may pass down on the right without extending behind the esophagus, but the left subclavian artery goes behind. This with the anterior location of the left common carotid artery constitutes an almost complete annular encirclement of the esoph-

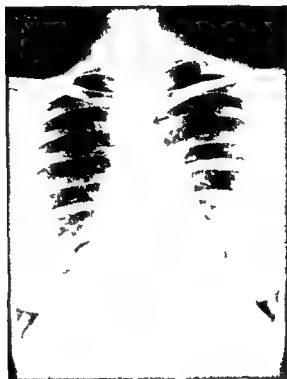
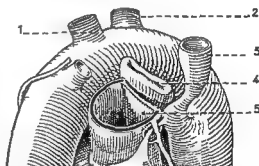


FIGURE 141 · Roentgen film showing right aortic arch pushing the esophagus to the left (arrow)

FIGURE 142 · Double aortic arch causing annular constriction 1 Right common carotid artery 2 innominate artery 3 left subclavian artery 4 esophagus 5 trachea (Case described by Pavlot Levrat and Guichard)



agus and trachea (Figs 140 and 141) The pattern is the exact opposite or mirror image of the classical case of *dysphagia lusoria* illustrated in Figure 137 In other cases there is a complete vascular ring surrounding these two organs In these there are two aortae, one usually the larger behind and the other smaller one in front In some instances the reverse of this is found Occasionally the anterior component of the ring is actually a patent ductus arteriosus or a ligamentum arteriosum (Fig 142)

Clinical Manifestations

Patients with an anomalous subclavian artery or a single aortic arch passing behind the esophagus rarely experience any symptoms and the condition may not be discovered except at operation or on performing an autopsy More and more these conditions are coming to light nowadays, however because of the

increasing utilization of angiocardiology as a diagnostic or prognostic measure in patients with thoracic disease. Occasionally they are discovered only during an operation involving exploration or dissection of the mediastinum. In a small percentage of these patients dysphagia may be troublesome, especially on swallowing large particles of food or as a result of too rapid eating.

On the other hand, those who have a nearly complete encirclement of the trachea and esophagus, and particularly those with a true double aortic arch comprising an annular constriction, may have disturbing symptoms. In many infants with this type of anomaly there is a predominance of respiratory symptoms with coughing, cyanosis, and suffocation during nursing. This is in part because of the compression of the soft posterior wall of the trachea by the distention of the esophagus which occurs during rapid swallowing, and in part because of the aspiration of regurgitated feedings. In some patients, however, dysphagia may not be noticed until they begin to eat a more solid diet, when the true nature of the condition will be discovered as a result of roentgen studies.

Diagnostic Studies

ROENTGEN EXAMINATION of patients with vascular anomalies causing compression of the esophagus is usually characteristic. When the aorta or right (sometimes left) subclavian artery passes behind the esophagus, a rounded in-



FIGURE 143 Deviation of the esophagus by a right aorta: dysphagia lusoria. Right anterior oblique projection (Jaubert de Beaujeu)

FIGURE 144 Roentgen film showing compression of the esophagus by a double aortic arch (see Fig 145)

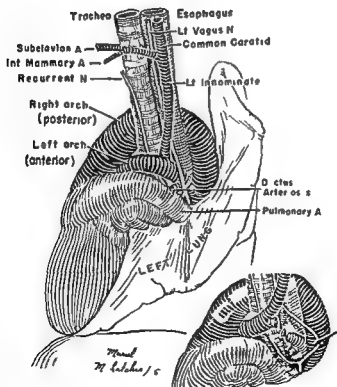


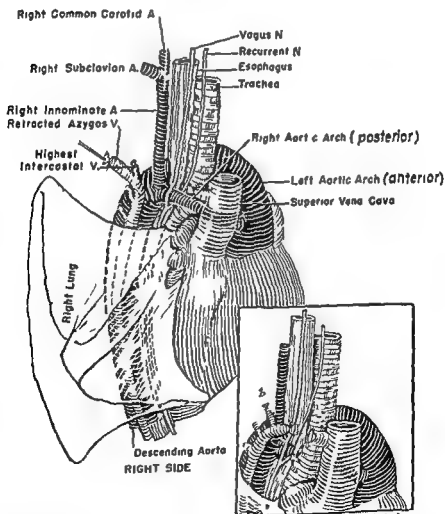
FIGURE 145 Drawing of the anatomical appearance at operation on the patient whose film appears in Figure 144. Insert shows release of constriction after division of the anterior vessel.

dentation is seen in the posterior wall of the esophagus, often with a forward deviation of the organ at that point (Fig 143). In patients with an annular double aortic arch a posterior higher and an anterior lower indentation are seen, as shown in Figure 144.

ESOPHAGOSCOPY is not indicated in the presence of any of these anomalies.

Treatment

If the symptoms are mild, control of the type of food ingested and correction of faulty eating habits may be all that is required. This is usually the case in patients who have a vessel passing behind the esophagus but without an annular constriction. If these measures fail, as is likely to be the case when there is a double aortic arch, the difficulty can be overcome by surgical division of the appropriate vessel to release the compression upon the esophagus (Fig 145, insert). In the usual instance the left side of the chest is chosen for the exposure of the vessel. This takes care of the occasional symptomatic case of right sub



Manual M. Katchen/46

FIGURE 146 Drawing of the anatomical appearance at operation of a double aortic arch which is the exact opposite or mirror image of that shown in Figure 145. Inset shows release of constriction after division of the anterior vessel.

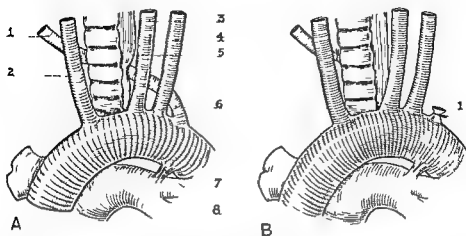


FIGURE 147 *A* Anatomical appearance at operation of a case of dysphagia lusoria with a right subclavian artery arising from the left of the arch and passing behind the esophagus 1 right subclavian artery 2 right common carotid artery 3 left common carotid artery, 4 left subclavian artery 5 compression of the esophagus by the anomalous right subclavian artery 6 origin of the anomalous artery 7 ductus arteriosus 8 pulmonary artery

B Appearance after division of the anomalous artery 1 ligated stump of the origin of the right subclavian artery

clavian artery arising from the left side of the aorta and the majority of instances of double aortic arch. In patients with a double aortic arch in whom the aorta descends in the right side instead of the left the anomaly can be reached more easily through the right side of the chest than through the left. In this instance the configuration of the constricting vascular ring is often anatomically the exact mirror image of the pattern of the vessels when the aorta descends on the left. An example is illustrated in Figure 146.

A midline sternotomy incision provides easy access to all of the anomalies of the aortic arch or its major branches thus eliminating the necessity for attempting to decide which side of the chest should be entered to provide the ideal exposure. The actual division of the offending vessel is accomplished readily between two ligatures (Figs 145 146 147).

No ill effects are experienced by the patient as a result of these procedures. With a double aortic arch the smaller of the two vessels usually in front, is always chosen for division leaving the true functioning aortic arch intact. In patients who have an anomalous subclavian artery there need be no fear for the blood supply of the arm. In fact because the vessel is divided close to its origin the collateral circulation is so efficient that after a few days the pulse usually reappears at the wrist.

Compression of the Esophagus by Acquired Abnormalities of the Aorta

Enlargement of the aorta from arteriosclerotic aortitis or more often aneurysms, whether syphilitic or arteriosclerotic may if severe cause a moderate degree of compression of the esophagus sufficient in extent to give rise to dysphagia. Usually it is involvement of the arch which gives rise to this condi-

tion, but an unusually large aneurysm of the descending aorta sometimes causes compression of the esophagus as well. So far as its effects upon the esophagus are concerned, the degree of dysphagia depends upon the size of the aneurysm causing the compression. The patient can usually swallow liquids or semisolids. Sometimes the severity of the dysphagia varies in a given case from time to time, depending upon the occurrence of reflex esophagospasm.

On rare occasions, enlargement and tortuosity of the lower thoracic aorta behind the point where the esophagus enters the hiatus in the diaphragm may cause enough pressure to induce dysphagia. The dysphagia is never pronounced although temporary lodgement of an incompletely masticated piece of meat may be experienced. Careful fluoroscopic observation is usually sufficient to establish the diagnosis. Esophagoscopic examination reveals no abnormality.

Diagnostic Studies

On ROENTGEN EXAMINATION it is usually the left wall of the esophagus which is pushed upon and the deviation is to the right and backward. This reduces the lumen of the organ sometimes to a semilunar shaped slit (Fig. 126).



FIGURE 148 Esophagoscopic view of the extent of obstruction due to compression of the esophagus by an aneurysm of the aorta

In some instances a pulsatile motion transmitted to the esophagus by the aneurysm may be recognized at fluoroscopy. When the aneurysm becomes partially obliterated with clots, this of course does not occur.

Although ESOPHAGOSCOPY is especially dangerous because of the possibility of inciting a rupture of the aneurysm by pressure of the end of the instrument, it often provides suggestive findings. Particularly in patients with dysphagia because of compression, an inward bulging of the wall of the esophagus is seen (Fig. 148). The mucosa on the side of the aneurysm appears hyperemic and abnormally vascular. Sometimes it is actually ecchymotic suggesting beginning leakage from the aneurysm.

Treatment

The treatment of aortic aneurysm is not a subject for consideration here. Obliteration of the sac may follow the introduction of large quantities of wire. A few successes are on record following surgical resection of the aneurysm with restoration of vascular continuity by insertion of an aortic homograft.

CHAPTER 8

Esophagospasm

Introduction

The term *esophagospasm* is employed to designate a hypertonic condition of the musculature of the organ which may be the result of various influences, either immediate or remote, or of causes which cannot be discerned. It is characterized by a temporary arrest in the progression of ingested material of variable duration, accompanied usually by an abnormal feeling of inability to swallow which is more or less painful. This definition, however, fails to include certain prolonged or even permanent spastic conditions. In fact the classic aspect of spasm, namely its temporary duration, does not always obtain. Examples of this will be discussed in their proper place.

Esophagospasm of unknown etiology is often given the term "essential." It should be recognized, however, that there is always a disturbance of the intrinsic neuromuscular mechanism of the esophagus or of the vegetative nervous system as a whole. More often it is secondary to an obvious lesion involving the esophageal mucosa, particularly esophagitis. This occurs more frequently than is usually recognized. In fact, esophagitis is the most frequent proximate cause of esophagospasm. A tiny fissure or a minimal ulceration in the phase of development may, in the beginning at least, be the trigger point which upsets the normal action of the neuromuscular mechanism. Once the lesion has become cicatrized or healed, the disturbance of the nervous system may persist perhaps as discrete lesions of the Auerbach plexus. Certain patients who are what one might call "spasmophilic" seem to have a predisposition to react by developing esophagospasm under the influence of the most minimal stimuli, whether physical or emotional, as during fear, worry, or grief. Thereafter, by a true vicious circle, the local disturbance of equilibrium can in turn accentuate the lesion which initiated it, chiefly by inducing esophagitis which in turn tends to upset the neural equilibrium still more.

If the condition persists, the problem becomes more complicated. The question to be decided is whether it is the prolongation of spasm which accentuates the various anatomical lesions or whether it is the latter which tend to

prolong the spasm. Furthermore, there is the question whether the mechanism is actually hypertonicity or whether there is a functional failure of the normal sphincteric mechanism of the pharyngo-esophageal segment or of the lower portion, as the case may be. Following the work of Hurst these disturbances are now thought of as being due to motor incoordination. The complexity of the problem increases as more is known about certain conditions such as the Plummer-Vinson syndrome, but until more is learned about these matters, the term "esophagospasm" will doubtless continue to be used.

From the anatomical point of view, esophagospasm may be subdivided according to the region involved. There is that occurring at the pharyngo-esophageal junction, that involving the majority of the thoracic segment, and that observed in the lower portion just above the diaphragm. Clinically speaking, it may be either transitory, acute, chronic, or even permanent.

Transitory and Acute Esophagospasm

ETIOLOGY: Rapid eating or drinking, insufficient mastication, and the ingestion of highly spiced foods are causes of esophagospasm which are common in the experience of almost everyone. In these a local or intrinsic neuromuscular reflex is involved. In other instances extrinsic reflexes may be a factor. For example, swelling and inflammation of the inferior turbinates of the nose, particularly in infants, has been found to cause spasmodic contraction of the pharyngo-esophageal junction and pharynx followed by serious difficulty in



FIGURE 149 Roentgen film showing reflex spasm of the lower esophageal segment (epicardial) secondary to a gastric ulcer (arrow)

swallowing both liquids and solids. This condition may be relieved by cocaineization of the turbinates or more permanently by electrocoagulation. In the lower esophagus reflex spasm is sometimes seen in cardiac decompensation and especially in coronary occlusion. Likewise viscerovisceral reflex stimulation is responsible for the occurrence of marked esophagospasm in patients with gastric ulcers, particularly those near the cardia and to a lesser degree in acute cholecystitis (Fig. 149). These reflexes are no doubt mediated through branches of the vagus nerves.

Esophagospasm of psychic origin is well known. It can be shown, for example, both radiologically and at endoscopy, that by inciting a patient to anger, esophagospasm may be initiated or aggravated if already present. Fear and worry are frequent causes in susceptible persons.

The condition may be observed also in patients with metabolic diseases such as diabetes, in Bright's disease, in rheumatoid arthritis, and in avitaminosis. The mechanism in these conditions is obscure but in beriberi, at least, pathological changes have been found in the vagus nerves.

Poisonous mushrooms and certain drugs such as atropine, nicotine, or arsenic when administered to excess may cause esophagospasm. The toxins elaborated by the invading organism or virus in tetanus and hydrophobia are classic examples of intoxication as a cause.

The importance of esophagitis as a local cause of esophagospasm has already been emphasized.

The symptoms and signs of transitory or acute esophagospasm vary, depending upon the region which is involved.

Spasm of the Pharyngo-esophageal Segment

CLINICAL CHARACTERISTICS. The pharyngo-esophageal segment is the region most frequently affected, particularly in neurotic subjects who are sensitive to the effects of emotional trauma or those of a hysterical temperament who tend to relieve their emotional conflicts by concentrating on the swallowing mechanism. Spasm in this region involves all the muscles of deglutition and would more correctly be called pharyngospasm. It is most frequently encountered in young women, eighteen to thirty years of age, who have a history of neurotic manifestations. It may be observed also merely as a result of rapid eating or in patients who because of ill-fitting dentures swallow their food in too coarse a form.

The attacks are intermittent and transitory, though of variable duration. They are often alarming to both the patient and bystanders. On the occasion of a fit of anger or the receipt of some emotional shock or as the result of an effort to swallow an insufficiently masticated bolus of food, deglutition is violently interrupted and succeeded by choking, retrosternal discomfort and oppression. Hawking, paroxysms of coughing, and efforts to vomit follow. At the same time the anguish of the patient is intense and his face becomes congested or even cyanotic. He grows agitated. Often he tries to swallow water but without success, the fluid regurgitating through the nose and mouth. Coughing becomes uncontrollable, the tears begin to flow, and abundant salivation follows, the result of esophagolacrimal and esophagosalar reflexes.

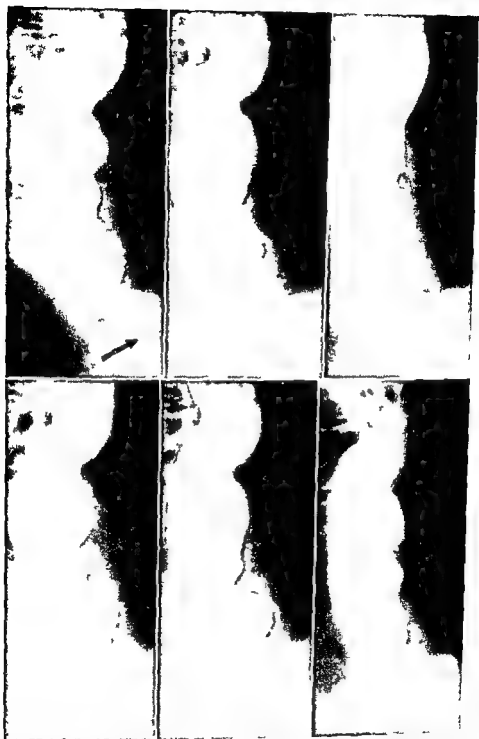


FIGURE 150 Serial roentgen films of the lower esophagus in a case of esophagospasm in the epicardial segment. Note the hypermotility of the organ proximal to the spastic area (arrow points to spastic epicardia which is seen in each of the films)

to force things by, finally give way to violent waves of antiperistalsis which lead to regurgitation and vomiting (Fig 150)

From time to time the esophagus contracts and shortens itself along a distance of as much as 10 to 12 cm. Then at the end of a variable period of time, depending upon the case, the opaque mixture penetrates into the stomach,

sometimes with a single ejaculation, sometimes in small spurts unless it is rejected in the course of vomiting efforts on the part of the patient. The examination of these patients can never be as carefully done as one would like, but serial films should be taken in various projections to avoid failure to recognize any abnormality of contour indicative of an underlying organic cause for the spasm.

On ESOPHAGOSCOPY the mucosa of the mediastinal portion usually appears normal or slightly hyperemic. As the lower end is reached, a tight stenosis is encountered. Here the mucosa is heaped up, congested, and sometimes even bloody and covered with residues of food (Fig. 151 and Plate III, 7). Sometimes all the food eaten may be piled up above the spastic area, requiring evacuation by aspiration or the use of a grasping forceps.

DIFFERENTIAL DIAGNOSIS. Without the aid of roentgen and esophagoscopic examinations the sudden arrest of deglutition and the clinical picture which accompanies it often lead to the suspicion that there is a foreign body lodged in the esophagus. Sudden paralysis of the pharyngeal musculature due to a vascular accident in the midbrain or the occurrence of a tabetic crisis must be thought of. In every older patient the diagnosis of an underlying carcinoma or a partial cicatricial stenosis resulting from esophagitis must be entertained. In fact, it must always be kept in mind that esophagospasm is frequently superimposed on a pre-existing organic lesion, including certain cases of achalasia as well.

The inciting cause of lower segment esophagospasm is not always obvious. It may be an over-large bolus of poorly masticated food or it may be a fit of emotional turmoil. Small inflammatory areas may be at fault. In this instance the stagnation of food with resulting local pressure on the walls of the esophagus gives rise to esophagitis which prolongs the tendency to spasticity. Sometimes even in so-called 'essential' esophagospasm a small foreign body hidden beneath a fold of mucosa may be discovered if a careful search is made. Often the site of lodgement of such an object is the upper esophagus, where its presence is obscured by associated pharyngo-esophageal spasm. The discovery and extraction of the foreign body may depend upon the use of a spatula for inspection or of a child's size esophagoscope.

When it comes to the use of ROENTGEN EXAMINATION to differentiate the possible organic causes, it is wise to use the erect posture in various positions and a barium mixture of the correct consistency. Serial films using a short

FIGURE 151 Drawing of esophagoscopic view obtained in a case of spasm of the lower esophagus at the cardia. Note the approximation of the walls of the esophagus. (After Guisez)

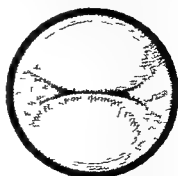




FIGURE 153 Roentgen film showing curling of the esophagus (man age 73)

ESOPHAGOSCOPY usually reveals that the esophagus is of normal length but often shows evidences of diffuse or localized esophagitis

TREATMENT is entirely symptomatic

2 The Barsony-Teschendorff Syndrome (Segmental Spasms)

Clinical recognition in a general way of a neuromuscular disturbance now known as the Barsony-Teschendorff syndrome was made many years ago. It was Barsony, however, among others who first called attention to the characteristic roentgenological appearance of the esophagus. He observed in two patients afflicted with ulcers of the duodenum that the esophagus displayed dilated segments separated by zones of constriction. He applied the term 'reflex dilatations' or relaxing diverticula to this appearance. It was chiefly Teschendorff, however, who elucidated the nature of the condition by demonstrating that the pearl necklace appearance of the barium filled esophagus disappeared an hour after the injection of atropine, only to reappear after the action of the antispasmodic agent had subsided.

The old idea that the appearance was due to mediastinal adhesions was thus disproved and subsequent autopsy studies as well as observations made at surgical operations have confirmed this by failing to disclose any periesophageal adhesions or evidence of mediastinitis. Furthermore, as Reich has shown the shadows are never quite the same at repeated examinations. All are now agreed that the condition is due to a disturbance of function without demonstrable organic changes. These areas of segmental contraction, however, are not always temporary deformities but may last many months.

ETIOLOGY As with other spastic states in the esophagus the patient usually is one predisposed to the development of the condition. Upon this predisposition are usually superimposed one or more functional physical or emotional initiating factors. Rapid eating, gluttony, insufficient mastication, the ingestion of highly seasoned, spicy, or coarse foods, too hot or too cold liquids, intense preoccupation with one's affairs, worry, fear, and the difficulties of existence are inciting causes in susceptible people. There is a sympathetic nervous system imbalance.

The age of the patient may vary from twenty-four to eighty years, with a maximum frequency from forty to sixty years. Rarely the syndrome may be observed in a spasmophilic infant. In this instance the condition is relatively simple as compared with adults. In infancy it is merely a manifestation of spasticity on the basis of a disturbance of function, whereas in adults the functional nature of the condition is often complicated by anatomical lesions, as with the other forms of esophagospasm.

There appears to be no sex predisposition.

The condition is frequently not recognized, or at least is misunderstood, in spite of its obvious characteristics. Given a person whose vegetative nervous system is susceptible, various secondary causes may produce this disturbance, as for example diseases of the gastrointestinal tract. Barsony observed eleven cases of segmental esophagospasm among seventy patients with duodenal ulcer. The occurrence may not be noticed, however, unless particular attention is paid to the esophagus in patients with duodenal ulcer. It is unusual in patients with gastric ulcers and in those with true diverticula of the esophagus. Disturbances of the gallbladder, particularly cholelithiasis, may be associated, but it is sometimes seen after cholecystectomy.

Cardiovascular disease, notably aortitis, left ventricular insufficiency, coronary arterial disease, and angina pectoris, may be inciting causes.

The occurrence of this condition in patients with Parkinson's disease, ovarian cysts, diabetes, renal lithiasis, and asthma can hardly be more than coincidence.

PATHOGENESIS Many theories of the pathogenesis of this abnormality have been advanced. There is currently fairly general agreement that there is a disturbance of the peristaltic activity of the esophagus. A plausible suggestion is that these segmental contractions are induced by localized tertiary waves of peristalsis in the lower half of the organ because of an imbalance with the primary wave. If the latter is strong and progresses with a normal intensity, the tertiary waves disappear and no evidence of spasm is seen. If the primary wave is feeble and fails to progress, the tertiary waves result in spasm. As a result of this basic mechanism the corkscrew appearance known as curling, with its irregular movements confined to the lower half of the esophagus and clearly distinguishable from the Barsony-Teschendorff syndrome, may result. Or the disturbance may take on the pearl necklace appearance characteristic of the syndrome. The roles of the swallowing center in the globus pallidum and of the vagus nerves in the production of the kinetic disturbance are not known.

Lortat Jacob has described an organic change in two patients with the Barsony-Teschendorff syndrome which he called diffuse nodular myomatosis.



FIGURE 154

FIGURE 155

FIGURE 154 Roentgen film showing tertiary contractions in a case of neoplastic stenosis of the lower esophagus

FIGURE 155 Tertiary contractions of the undulatory type (case of Type II achalasia)

At operation he found multiple thickenings of the muscular coat occurring at intervals of several centimeters. The nature of the nodularities was demonstrated histologically, and the condition was shown not to be a case of multiple myomata of a neoplastic nature.

Examples of segmental spasm are shown in Figures 154, 155, 156, 157, and 158. The difference between this segmental contraction and the corkscrew appearance in 'curling' (Fig 153) is obvious.

CLINICAL CHARACTERISTICS Dysphagia is intermittent and capricious. It usually occurs suddenly and is often complete. In the beginning this stoppage gives way to a fit of suffocation. Those who have had previous attacks, however, know what to expect, they swallow water, rub the epigastrium or apply hot compresses over the chest wherever they think it might help.

The paroxysm is usually self-limited. Sometimes it may last several minutes only, sometimes several days or even weeks. In some instances a gastrostomy may become necessary.

The attacks occur at variable intervals, sometimes close together, often as infrequently as once or twice a year. Sometimes the trouble is confined to meal-times and at all other times the physiology of the alimentary tract is normal. The character of the food may be a factor as well.

Regurgitations of mucus and saliva are usually not abundant. They occur at the time of the dysphagia, sometimes only at the end of an attack and sometimes not at all.

Salivation is more or less active

The dysphagia is usually accompanied by pain but pain may be lacking. The location of the discomfort varies. It may be beneath the sternum or the xiphoid process in the epigastrium in the right or left hypochondrium, or in the back. Radiation of the pain is upward to the neck, face and ears or to the back and shoulders. The intensity of the pain varies with the patient. In some it is intolerable, like the cutting of a knife or like the anguish of angina pectoris. Others experience a sort of hunger pain like that of duodenal ulcer. A condition which may coexist with this form of esophagospasm as has been mentioned before. In all some form of sensation of oppression or constriction of the chest is felt. Still others experience pyrosis, dyspeptic discomfort in the region of the stomach, eructations of air and sometimes headache. The symptomatology is profuse and varies with the patient.

As with all forms of spasm of the digestive tract the outstanding characteristics are violence of onset, capricious evolution and sudden termination of the attack.

ROENTGEN EXAMINATION. Examination of patients with segmental spasm of the esophagus often demands special attention to technical details and prolonged and careful observation. Both the erect and the recumbent positions should be used and the Trendelenburg maneuver may be required. Sometimes at the start of the examination the esophagus appears to be normal but after several deglutitions, often with the smallest mouthful the characteristic shadow appears. A thick, creamy barium mixture is recommended. Films taken in series are important.

The appearance varies. There are two principal patterns. In the one exaggerated waves of peristalsis segment the margin of the esophagus so that its shadow looks like a string of beads but the descent of the opaque mixture is

FIGURE 156 Roentgen appearance of the Barsony Teschendorff syndrome representing marked segmental contractions in a case of ulcer. Note the superimposed shadows simulating diverticula (pseudodiverticular type)



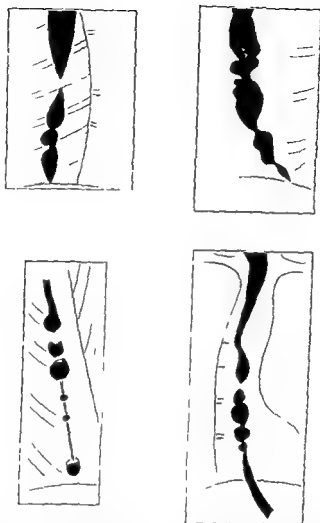


FIGURE 157 Diagrams copied from roentgen films showing examples of segmental esophagospasm Barsony, Teschendorff syndrome (Diagrams of Chêne Schmidt Panhuseyn and Palugyay)

rapid The contractile aspect of the waves is accentuated The esophagus is in fact hyperkinetic

In the other, the peristaltic waves remain exaggerated but the mixture does not descend The contractions are held in one place The esophagus is truncated by circumferential constrictions between which its walls become dilated, giving the appearance of a series of balls, one upon the other with a string between (Fig 156) This dilatation, however, is often irregular or eccentric There are appearances of semicircles, rounded images and elongations in various directions either to the right or to the left Some of the shadows are shaped like a hat (Figs 157, 158) The volume of each shadow varies

Besides these two chief patterns, other forms of behavior may be noticed In one patient the barium shadow may rise and fall in the esophagus like an elevator In another the abnormality may appear at the first swallow then disappear to be followed in several minutes by marked hyperkinesia of the esophagus with segmentation into any of the forms described above Shadows representing the segmentations tend to appear slowly and the rate of passage of the

barium varies. Sometimes after a more or less prolonged interval the esophagus empties and its contour becomes normal. Sometimes the image does not change but the opaque mixture descends slowly looking like a snake as it progresses.

One gets the impression that the esophagus has a predominating tendency to contract but without normal coordination of the intrinsic neuromuscular mechanism. It forms bellies and knots. The former have been wrongly spoken of as diverticula. Characteristically, however, the pattern does not remain permanent (Fig 158). The shadows vary in size and shape from patient to patient and from time to time.

The extent of the involvement of the esophagus varies but in about 65 per cent of the patients only the lower third is affected. In a few the condition may be localized to the middle third and in exceptional cases the entire organ may be involved.

As mentioned before, the contractions can be eliminated by the administration of atropine. About forty five minutes are usually required for their disappearance but with the usual dosage of 0.5 mg they return after about two hours. Morphine and amyl nitrite also sometimes abolish the contractions. In some patients, however, the administration of antispasmodic and relaxing drugs produces no effect.

ESOPHAGOSCOPY. As with all spastic conditions of the esophagus the examination may be difficult. Adequate preliminary sedation and liberal use of cocaine and Adrenalin solution topically are essential. Visual control and slow manipulation must be observed. The mucous membrane appears normal in the

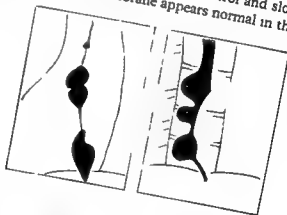
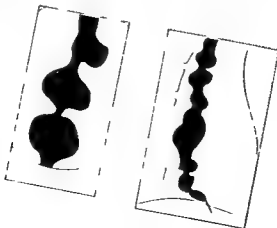


FIGURE 158 Diagrams copied from roentgen films showing examples of segmental esophagospasm Barsony Teschen dorff syndrome (Diagrams of Chene Schmidt Panhuseyn and Palugayay)



vast majority of the patients. In a few, esophagitis or ulceration may be found just above the cardia. Sometimes the passage of the instrument evokes the intense pain of acute esophagospasm.

DIFFERENTIAL DIAGNOSIS The usual causes of dysphagia must be considered. Organic lesions such as carcinoma may have to be excluded by esophagoscopy. Sometimes angina pectoris is wrongly diagnosed in these patients. The diagnosis cannot actually be established without careful roentgenological investigation. The success of this examination depends entirely upon the alertness of the roentgenologist. If he is not familiar with the Barsony-Teschendorff syndrome, he may come to the erroneous conclusion that the patient has stenosis with multiple areas of constriction, or that there are multiple diverticula. The opinion is sometimes given that the esophagus is surrounded by mediastinal adhesions.

In every case the patient should be studied to discover any possible underlying cause for the phenomenon, such as esophagitis or esophageal ulcer. The frequency of coexisting duodenal or gastric ulcer has been mentioned. A carcinoma of the stomach may be found. Biliary tract disease should be looked for. If no local inciting cause is found in the gastrointestinal tract, various other studies should be made. Spasm of this type may be the reflex manifestation of an organic affection of some other system, such as the cardiovascular apparatus or the female pelvic organs. It should be reiterated, however, that the patient is always an individual with an unstable sympathetic nervous system. The condition, however, cannot be brought on at will nor does it always occur in patients with esophageal inflammation, much less in those with duodenal ulcers or affection of other organs. If all examinations are negative, we must fall back upon the term "idiopathic," which does nothing but mask our ignorance.

PROGNOSIS The outcome of the case depends upon whether or not a contributing cause which can be eliminated by suitable treatment is found. If such is the case, the cure of the duodenal ulcer, esophagitis, or gallbladder disease often brings about a disappearance of the esophagospasm. In cardiovascular disease, however, the results of treatment may be only partially successful, with failure to relieve the reflex esophagospasm in some instances. In those in which no underlying organic cause exists, the disturbance may last from a few months to fifty years or more. The disease is not in itself lethal. The usual course is benign. In cases where a gastrostomy has been necessary because of the relentless nature of the spasm, the condition usually subsides, possibly because of the relief from the necessity for swallowing anything but the saliva. The gastrostomy can then be abandoned after the passage of a variable period of time.

TREATMENT No comment is needed about the treatment of organic lesions which may be the inciting cause of this form of esophagospasm. Esophagitis is treated by dietary regulation and the use of demulcents and antacids. When no inciting cause is found and in other cases as temporary palliation, recourse is usually had to antispasmodic medication as with atropine or Banthine, along with sedatives of the barbiturate group or relaxing drugs such as papaverine.

Splanchnic block is not of much value.

Sometimes the spasm disappears after one esophagoscopy, a result which confirms the capricious nature of the disturbance. This may mean, of course that there is a large psychic element in this bizarre form of esophageal dyskinesia.

Dilatation with ordinary bougies or with the mercury bougie may be tried, often with success.

Surgical interference other than the performance of a gastrostomy is practiced only in the most recalcitrant cases. The Heller operation (esophagomyotomy) may be beneficial, but it must be done according to the Fontaine technique, with an incision through the muscle at least 8 to 10 cm. in length. This operation, it should be recalled, not only divides the muscle fibers but to some extent interrupts the sympathetic and vagus innervation of the involved segment as well.

Sympathetic ganglionectomy produces no relief.

Chronic Esophagospasm

Persistent spasm of the esophagus may occur either at the cricopharyngeus muscle or at the lower end. Actually the term *permanent occlusion* is more exact than persistent spasm. In fact, the closure of the superior orifice and inferior segment of the esophagus upon the arrival of the alimentary bolus can be the consequence of a defect of coordination as much as an active spastic contraction. If spasm is at fault, it is not essential to admit that it is really permanent.

Sphincteric hypertonicity and motor incoordination can be set in motion only at the moment when in its descent through the hypopharynx and upper esophagus the alimentary bolus reaches the segment of mucosa from which the sphincter-regulating reflex arises. Then either the regulating reflex is not aroused or it ends in an occlusion resembling that which normally protects the esophagus or the stomach against the entry of a voluminous bolus or one composed of irritating or insufficiently masticated food. However that may be the permanence of the action provokes dilatation of the segment above the sphincter or contracted segment the penetration of which becomes irregular or ineffective. This phenomenon is common to all parts of the digestive tract.

Persistent Spasm of the Cricopharyngeus Muscle

ETIOLOGY. Montadon who has given the problem special study based upon the observation of a series of patients proposes a classification into two types. One is a *dynamic* or *neuromuscular form* in which nerve lesions involving the ganglionic plexus predominate. This is characterized clinically by the intermittency of the paroxysms of contraction at first, which become progressively worse as time goes on and finally end with permanent painful contracture of the cricopharyngeus muscle as described by Negus. The other is a *passive, myopathic* or *interstitial type* in which diffuse interstitial fibrosis or degenerative myositis is found in the muscle. In these the lack of elasticity and contractility of the sphincter gives rise to a painless progressive dysphagia with delay in the ingestion of solids at first soon giving way to complete and irreversible obstruction. The first is a persistent spasm the latter involves an organic change in the muscle. In the latter infiltration with lymphocytes and degeneration and fibrosis



FIGURE 162 Myopathic occlusion of the mouth of the esophagus. Section from the cricopharyngeus muscle. In the periphery are shown muscle fibers which retain their myoglobin. In the center the atrophy is almost complete. At A is seen fibrous tissue substitution of the musculature without evidences of inflammation. (After A. Montadon and E. Rutishauser.)

chronic myopathic spasm of the mouth of the esophagus may be one (Figs 159, 160, 161, 162)

CLINICAL CHARACTERISTICS Patients who develop this condition give a history of previous acute paroxysms of spasm. They are usually neurotic in tendency, almost always elderly, and are often gluttonous, rapid eaters with defective teeth or dentures. More rarely the condition may appear out of a clear sky without antecedent attacks and present itself as a rapidly progressing dysphagia.

In the usual case the patient complains of a hindrance or an abnormality of deglutition. From time to time he experiences a choking sensation at meals, especially when attempting to swallow bread or meat. If the mouthful is too large, it provokes an acute embarrassment with painful swelling of the base of the neck. The patient often executes repeated movements of flexion or rotation of the head in an effort to force the material down. Sometimes the food is ejected forcefully through the mouth and nose. Liquids in small amounts pass more readily.

Gradually, eating becomes more difficult and more painful. The periods of

comfort between attacks diminish. Little by little the amount of food swallowed at one time must be diminished, and repeated efforts to swallow are required to get even small quantities down. Finally, after several attempts, a unilateral or bilateral swelling may develop in the base of the neck, the result of dilatation of the hypopharynx.

In the beginning the trouble is purely functional, but at last anatomical changes ensue. Diffuse dilatation of the pharynx may occur, or a diverticulum may develop. From then on, the anatomical changes predominate and the cricopharyngeus muscle itself undergoes a permanent alteration.

ROENTGEN EXAMINATION reveals a stoppage of the barium just above the cricopharyngeus muscle. Passage through the sphincter may be completely interrupted or may take place slowly, drop by drop. In certain patients the barium outlines a more or less clearly shown, regular, midline or lateral pocket which corresponds to the dilatation of the hypopharynx or an actual diverticulum. The nature of the condition can be discerned by a study of the roentgen films.

ESOPHAGOSCOPY yields variable results. In the beginning the mouth of the esophagus, even after adequate use of cocaine and Adrenalin, is slow to open and fails to yield without steady pressure. An almost imperceptible cleft with its concavity anterior is noticed as a result of the bulging of the muscle layers proximal to the sphincter.

At other times in spite of anesthesia the tube provokes contraction and the cricopharyngeal pinchcock appears like a jutting collar beneath the mucosa. Steady continuous pressure with the end of the instrument, aided by the application of a pledget of cotton soaked in cocaine solution heavily fortified with Adrenalin, is the only way to overcome the contracture. The cervical esophagus, as it unfolds little by little in front of the instrument, gives the appearance of a rosette.

In patients with dilatation of the hypopharynx, lateral motion of the tube is easily accomplished. The mucosa is swollen and hyperemic, often covered with particles of food. Sometimes foci of leucoplakia are seen. The mouth of the esophagus is hard to find and often it can be passed only with the aid of a bougie as a guide (Chapter 4). The passage of a bougie imparts information which varies from one time to another. Sometimes the instrument is held up 15 cm. from the dental arch, sometimes after a temporary arrest it descends with more or less ease. Its introduction is facilitated by repeated swallowing efforts. The variability of the degree and duration of the obstruction is an important diagnostic point.

If carcinoma is suspected, in efforts to make a definite diagnosis one should be reminded of the dangers of performing a biopsy in this region, a procedure which should be restricted to the cases in which there is a protruding mass. Cytological studies of secretions are safer (see Chapter 27).

Persistent Spasm of the Lower Esophagus ("Giant Muscular Hypertrophy")

An unusual abnormality of the lower esophagus which bears a striking similarity to the irreversible changes in the cricopharyngeus muscle following long continued spasm has been encountered. This is characterized by a tremendous thickening of the muscular coat, chiefly the circular layer which in one



FIGURE 163 Giant muscle hypertrophy of the esophagus. View obtained at operation for the relief of dysphagia accomplished by esophagomyotomy. Note enormous thickening of the muscular wall. Intact mucosa is shown at the bottom of the incision between the two muscle surfaces (chronic spasm).

patient was 8 mm in thickness (Fig. 163). There is no evidence of acute inflammation, but a moderate degree of infiltration with round cells is seen. On palpation the abnormal segment of esophagus is firm but does not have the rigidity of a carcinoma. The longitudinal extent of the condition varies, but the lower one-third to one-half of the organ may be involved. The proximal end of the abnormal thickening can be felt as a firm edge, suggesting that there is an abrupt change from pathological to normal muscle. The lower extent of the thickened segment is at the cardia.

CLINICAL CHARACTERISTICS. Patients with this peculiar abnormality have a long history of intermittent dysphagia often associated with emotional disturbances suggesting the occurrence of attacks of lower segment esophagospasm. As the attacks of dysphagia become more severe and more persistent, it is more and more difficult for the patient to eat a normal diet. Gradually the obstruction becomes almost complete, ending with a permanent inability to swallow anything but liquids. Substernal pain with radiation to the back, shoulders, and neck may be felt. Pyrosis may be troublesome.

The ROENTGEN APPEARANCE of chronic spasm or muscular hypertrophy of the lower esophagus is characterized by delay in the passage of barium into the stomach but without obvious narrowing of the abnormal segment except at the lower end (Fig. 164). The peristalsis tends to be hyperactive. In two patients observed recently, the proximal edge of the hypertrophied spastic portion was sharply defined like a shelf. This may lead to the erroneous diagnosis of carcinoma in these patients who might otherwise be assumed to have achalasia (Type 2) (see Chapter 9).

FIGURE 164 : Roentgen film of the esophagus shown in Figure 163 esophagogram made before operation Woman age 70



It should be pointed out that the conception that esophagospasm, especially of the chronic or permanent form is more organic than functional has been neglected. Anatomical, pathological and clinical studies combined are needed to overcome our ignorance of this condition, the functional aspect of which until now has been overemphasized. Chevalier Jackson has always rightly insisted upon the importance of the search for an organic cause.

TREATMENT The treatment of chronic spasm of the cricopharyngeus muscle, a condition which ends with a permanent organic change in the muscle itself, is unsatisfactory. Relaxing agents are valueless. Dilatation is either impossible or of temporary benefit only. Efforts to relieve the constriction by surgical division of the muscle in the midline posteriorly in the manner of a Heller or Ramstedt operation are disappointing. In some instances final recourse must be had to the performance of a gastrostomy for the purposes of alimentation and hydration.

In persistent spasm of the lower esophagus (giant muscular hypertrophy) the only successful treatment may be resection of the diseased segment. Relief is dramatic and permanent. An unusually long esophagomyotomy may be tried if the longitudinal extent of the abnormally thickened indurated muscle layer is not too great.

Esophagospasm in Infants and Children

Intermittent Dysphagia in Infants

Although esophagospasm seems to be a rare occurrence in infancy, spasm at the mouth of the esophagus is sometimes observed in bulimic infants. When it occurs at more or less prolonged intervals this spasm can provoke both in infants and in older children true paroxysms of intermittent dysphagia. As with adults, permanent dysphagia may supervene if these attacks persist.

DISEASES OF THE ESOPHAGUS

CLINICAL CHARACTERISTICS In the newborn, intermittent dysphagia is usually observed during the first few days of life or in the first few weeks at least, when the child suddenly begins to regurgitate its feedings. The distinction between regurgitation and vomiting is easily made, the food is rejected rapidly after ingestion, without effort and without evidence of nausea. The material which is regurgitated is mixed with mucus, is odorless, and shows no sign of fermentation. Clinical examination reveals no evidence of activity of gastric juices. The rejection often takes place during sleep. As time goes on, a phase of absolute intolerance of food is reached. This is due to spasm superimposed upon inflammatory changes and swallowing becomes impossible.

During this critical period the growth of the infant is retarded, the skin becomes dry and wrinkled, the urine becomes scanty, and the weight diminishes rapidly, partially because of dehydration.

Often the attacks are transitory, recurring at variable intervals with intervening periods of complete remission. Thus there is established a cycle alternating between apparent good health and troublesome digestive disturbances. This cycle may last for months or years.

ROENTGEN EXAMINATION makes possible identification of the location of the spasm. A thorough familiarity with the behavior of the esophagus in infants, as contrasted with that in older children and adults, is essential in order to interpret the findings. After the second year these differences gradually disappear.

PASSAGE OF A BOUGIE carefully done may provide information of the first importance. One should begin with a soft bougie of small diameter. Once the pharynx has been passed, the instrument may be stopped by spasm of the cricopharyngeus muscle. With a little patience and a wait of several seconds, this usually can be overcome. On reaching the cardia, additional resistance may also be found. Once again after several minutes of firm, gentle pressure the bougie will pass, imparting as it does a characteristic sensation to the guiding hand. To the experienced operator this maneuver often assists in making the diagnosis of spasm as opposed to organic stenosis.

ESOPHAGOSCOPY With a small infant in dorsal decubitus, the instrument can usually be introduced readily with slight pressure under visual control. The mucosa of the entire thoracic portion of the esophagus appears pink, and the normal movements synchronous with those of respiration and of the heart beat can be seen. The cardia presents its normal infundibular appearance and under the pressure of the esophagoscope the spasm yields more often than not without any evidence of pathological changes in the mucous membrane. This so called negative examination, however, is worth while as it once again excludes organic pathology.

DIFFERENTIAL DIAGNOSIS Esophagospasm in infants must be differentiated from the ordinary regurgitation which results from too rapid nursing and resultant overloading of the stomach with milk or from too frequent feeding. This condition is easily distinguished from esophagospasm by the fact that the milk is curdled showing that it has been at least a short time in the stomach. Differentiation becomes easy after regulation of the feeding program. If the regurgitation persists, indigestion from improper feeding mixtures is excluded.

by observing the stools, which are usually granular in appearance greenish in color, acid, and irritating. The buttocks are often reddened and sore. The disturbance may result from an excess of fat. The child is often pale and pasty in appearance. In some instances also, vomiting may result from inadequate feeding because of the hunger of the infant and its eagerness to be fed. The thin appearance, constipation, and behavior of the child are characteristic. In other infants, aerophagia may be a factor in excessive vomiting because of the contraction of an overdistended stomach.

Pyloric stenosis must be excluded also. The vomiting in this condition may be prompt, often following immediately after nursing. The attack is, however, preceded by paroxysms of pain and the vomiting is explosive (projectile), unlike the regurgitation of esophagospasm. Furthermore, once again, the milk is curdled. Examination of the child's abdomen reveals visible peristaltic waves which subside after the vomiting is over. The enlarged pyloric muscle sometimes can be felt.

Other conditions such as congenital stenosis, esophageal webs, and atresia with tracheo-esophageal fistula have been discussed in Chapter 6.

Esophagospasm in Older Children

As in infants, esophagospasm may appear suddenly, but in the older children almost always there is a history of previous mild attacks. The situation is similar in its etiology and manifestations to that which prevails in adults. There is undoubtedly a constitutional predisposition with hyperreactivity of the nervous system accompanied by hyperexcitability of the smooth muscle layers of the digestive tract localized in these patients to the esophagus. In other words the patients are spasmophilic.

There is often, however, a local or inciting cause varying, as the case may be. Sometimes there is an anatomical abnormality, sometimes a minor pathological change such as esophagitis. A local functional abnormality may exist but there is always the fact that it alone, without added neuromuscular disturbances, is insufficient justification for the disturbances which are observed. In some instances, regurgitation of acid gastric secretions into the lower esophagus may be an inciting cause. The condition appears to be especially prone to occur in children with hereditary syphilis.

CLINICAL MANIFESTATIONS As in infants, there is not usually actual vomiting. The condition is characterized by the occurrence of effortless regurgitation which develops rapidly after eating and is unattended by nausea. The attacks are typically capricious as to frequency, with great variations in the lapse of time between them. Sometimes the dysphagia is more marked with solids, sometimes with liquids. The attacks occur in paroxysms which, as the child grows older, may diminish in intensity and frequency of occurrence.

As with the infant, however, the spasm of the esophagus may evolve into a more serious form, ending sometimes in a state of pronounced physiological disturbance if prompt and appropriate treatment is not begun.

If the paroxysms are oft repeated, a state of veritable permanent dysphagia may supervene. This form of the disease, which is never observed in infants, is encountered only in older children. The regurgitation becomes a daily occur-

rence No food may be taken without being rejected immediately. The child becomes emaciated and his condition grows more and more serious as the intolerance of his esophagus increases.

ROENTGEN EXAMINATION after the ingestion of barium mixture shows an enlarged esophagus ending in a fusiform shadow just above the cardia, beyond which nothing appears to pass.

At **ESOPHAGOSCOPY** the instrument enters the mouth of the esophagus easily and, once the cervical region is passed, the appearance is that of abnormal dilatation. The lumen is usually full of the debris of previously eaten food. The walls of the esophagus are often macerated. At the cardia the esophagus is pinched together, the mucosa producing a circular heaped up appearance. The orifice is closed and often nothing but a filiform bougie can be passed through it.

DIFFERENTIAL DIAGNOSIS The condition can be distinguished usually by the typical clinical manifestations and particularly by the roentgenological and esophagoscopic findings. Although as in adults the condition may affect the mouth of the esophagus or more rarely the main portion, it is usually confined in children to the lower end. For this reason it is often difficult to distinguish esophagospasm from mega esophagus which, though it may occur, is less frequently observed in children. The roentgen appearance may assist in making the differentiation. The gradual onset of the dysphagia in mega esophagus is also a differentiating characteristic. The lack of any real element of spasm in many instances of the latter condition is also an outstanding feature to be described elsewhere (Chapter 9). Other disorders have already been mentioned in discussing the diagnosis in infants and adults.

TREATMENT In infants and children afflicted with transitory intermittent esophagospasm, treatment should be directed toward improvement of the general condition. This is important because of the obvious fact that there is invariably a special sensibility of the nervous system. Antispasmodic medications are useful but one should always search for a local inciting cause. This may actually be a physiological disturbance which without any predisposition of the nervous system would be insufficient to bring on an attack of spasm.

An esophagoscopy should be performed at least in the beginning to exclude organic lesions. Its use, furthermore, may be followed by improvement in the symptoms. The same is true with bougienage, which should be tried if the condition persists. The bougie must be passed as always under visual control to avoid perforation. It should be left in place as long as possible. In the child, frequent repetition of this treatment is rarely required. The relief obtained is usually rapid if not immediate. After two or three dilatations the child swallows better and more easily.

The condition is, however, subject to recurrences and the dilatation may have to be repeated from time to time, beginning in each instance with small sizes and increasing progressively. This treatment can be applied at an early age. Chevalier Jackson reported a case of a newborn, two days old, who could not nurse or swallow. Esophagoscopy abolished the spasm in this child. Endoscopy alone may be sufficient to overcome the difficulty even in older infants.

CHAPTER 9

Mega-Esophagus (Achalasia)

THE TERM *mega-esophagus* is applied to a peculiar condition of unknown etiology characterized by a striking degree of dilatation and muscular hypertrophy of the major part of the esophagus above a constricted often atrophied distal segment. Depending upon the innumerable theories of its origin, the disease has acquired various other designations such as 'idiopathic dilatation', 'congenital dilatation', 'cardiospasm', 'phrenocardiospasm', 'cardio esophageal constriction', 'essential constriction of the lower esophagus', 'esophagospasm' and 'achalasia'. None of these is entirely satisfactory although the term *achalasia*, proposed by Hurst, is more accurately descriptive of the abnormal physiological behavior of the esophagus in this disease.

The term *mega-esophagus* is useful because its universal applicability makes it understood by all concerned. The more frequently employed designation 'cardiospasm' should be abandoned because the seat of the disturbance is not at the cardia and because spasm plays at best a secondary role in the manifestations of the disease.

Above the abnormal lower segment the circumferential dilatation involves almost the entire esophagus and in some instances there is also considerable elongation as well which leads to a marked tortuosity of the organ. There are two distinct anatomical types which can be recognized both by the clinical manifestations and by the appearance at roentgen examination as will be shown.

The disease was first described in 1821 by Purton who based his observations upon an autopsy examination. In recent years, much has been learned by roentgen examination, esophagoscopy, and inspection of the esophagus at operations performed for the relief of the dysphagia.

Etiology and Pathogenesis

No proven cause for mega esophagus has been found. It is relatively common in occurrence, being second in frequency to carcinoma. It tends to be more prevalent in certain parts of the world, notably in the São Paulo province



FIGURE 165 Spasm of the epicardial segment of the esophagus in an infant. Dilatation of the esophagus above (achalasia).



FIGURE 166 Achalasia in a two-year-old child treated by esophagoplasty. This child had experienced dysphagia for 18 months or more preceding operation.

of Brazil where it is found in a high percentage of the Indians. The diet of these people consists chiefly of corn (maize), a food which is mostly carbohydrate and low in protein and vitamin content.

The disease shows no predilection for either sex.

It may be observed in childhood, even in infancy but it is characteristically discovered either in adolescence or more often in adult life. Cases are on record, however, of patients with the disease varying in age from a few months to seventy five years. In fact, it is always difficult to determine at just what age it actually began. Figures 165, 166 and 175 are examples of the roentgen appearances of the disease at the extremes of life. Figure 167 shows the actual anatomical appearance of a Type 2 achalasia in an infant.

In many instances it appears that the patients afflicted with mega esophagus have a neurotic temperament or are at least emotionally unstable. Sometimes the onset of symptoms coincides with the occurrence of an emotional shock like the death of a dear relative. It should not be forgotten, however, that the frustrations and embarrassments attending the clinical manifestations of the condition may in themselves be responsible for emotional disturbances or changes in personality in otherwise normally adjusted persons.

Numerous attempts have been made by a variety of authors from many countries to discover some physical cause. External trauma, the local effects of coarse foods or very hot or very cold liquids, infectious diseases such as scarlet fever or measles, syphilis, tuberculosis, neurotropic toxic substances, tobacco, alcohol, endocrine imbalance, metabolic disturbances, and many others have been mentioned. Lack of certain vitamins, notably the B complex, as in the diet of the Brazilian natives, may be a factor under particular circumstances of poor diet or faulty absorption. No factual knowledge, however, is available.

Before the days of surgical treatment there was talk of periesophageal adhesions, or pressure against the lower esophagus by the liver or spleen or the margins of the esophageal hiatus of the diaphragm. Since at surgical operations none of these supposed abnormalities has ever been found, these suppositions have been abandoned. Periesophageal inflammation and adhesions, however, may develop as the result of injury to the narrow lower segment from forceful

FIGURE 167 Drawing of the esophagus of an infant with typical mega esophagus (achalasia)



dilatation or actual perforation Esophagitis unprovoked by mechanical trauma is actually rare in this disease

The coexistence of a gastric ulcer or of acute gastritis has sometimes led to the assumption that the mega-esophagus in such a patient had developed on the basis of some sort of reflex stimulus from these foci Since it has become obvious, however, that a relatively high percentage of patients with achalasia of the esophagus have an associated delayed emptying of the stomach due to dysfunction of the pylorus, this correlation should be abandoned In patients with gastric retention, gastritis and gastric ulcers are no doubt the result of hyperacidity and excess pepsin secretion caused by antral hormonal stimulation as Dragstedt has shown They are the complications of the underlying functional disturbance of the upper gastrointestinal tract rather than its cause Figure 168 illustrates the evolution of a case of mega-esophagus following the development of a gastric ulcer at the age of fifty-two years Changes in the roentgen appearance of the esophagus over the course of nine years are shown

All of this points to the inadequacy of any explanation of the disease in the vast majority of the cases except as a disturbance of the neuromuscular mechanism of the esophagus It should be emphasized, however, that the abnormality of the lower segment as observed at operation, contrary to what might be expected as a result of roentgenological and esophagosopic appearances, gives no evidence, with the one exception to be described, of the presence of spasm In the abnormal segment the muscle is usually thinned out and atrophic in appearance, and the total diameter of the organ there is usually greatly diminished Spasm of the muscular coats of the lower esophagus must therefore be eliminated as a cause of the disease in the majority of instances Furthermore, inasmuch as the cardiac orifice is always normal in size and not ever surrounded by hypertrophied muscle layers, cardiospasm can be excluded as a cause of the proximal dilatation

It is not at all clear whether the alterations characteristic of this condition are always due to a single cause It appears that mega-esophagus is not a distinct disease in itself It is more likely a syndrome resulting from diverse causes whether congenital or acquired, which act upon the nervous system at some level, either the cerebral centers, the spinal cord, the peripheral nerves, or directly upon the myoneural junction to produce disturbances of the intrinsic innervation of the esophageal musculature

Contrary to the conception that spasm is the inciting cause, Zenker and Ziemssen have suggested that the condition is the result of atony of the musculature of the esophagus That a relative degree of atony exists cannot be denied, but atony is not the entire cause of the difficulty It is more likely an accessory factor depending upon the basic disturbances of innervation

The most plausible explanation is the theory of Hurst and of Etzel who believe that mega-esophagus is due to a disorder of the emptying mechanism of the lower esophageal segment It was Hurst who introduced the term 'achalasia' to represent his hypothesis regarding the nature of the neuromuscular disturbance in this disease It should be recalled that in the normal esophagus, as the mouth of the organ closes during the deglutition of food or liquid, there is a reflex relaxation of the lower segment which makes way for the ingested

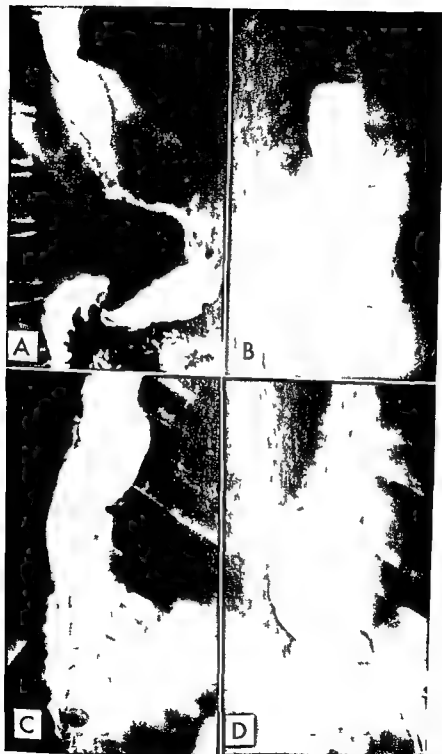


FIGURE 168 Series of roentgen films showing the evolution of a case of achalasia A Appearance in 1942 B 1946 C 1948 D 1951



FIGURE 169 : Mega-esophagus Upper third of the esophagus showing signs of chronic inflammation in Auerbach's plexus numerous newly formed vessels and lymphohistiocytic cells The arrow points to the remains of a ganglion cell (E Etzel)

material which is propelled there by the waves of peristalsis of the middle portion, aided of course by the effects of gravity. The vagus nerves through the intermediary of the recurrent nerves assure the contraction of the mouth of the esophagus. The opening of the lower esophageal segment depends upon excitation of the vagus nerves, and the closure upon the action of the sympathetic innervation upon a center of contraction or tonicity there. The theory of Hurst is that mega-esophagus results from a lack of the normal co-ordination between the mouth of the esophagus and the sphincter like lower segment which leads to a failure of the latter to open at the proper moment. To this phenomenon the name 'achalasia' was given.

This theory presupposes abnormalities of the nervous system which are now known to occur. Inflammatory changes have been described in the intramural plexuses of Auerbach and Meissner. These changes vary from simple leucocytic infiltration and more or less intense congestion to complete degeneration and ultimate fibrosis of the plexuses, particularly their ganglion cells (Figs 169, 170-171). Pigmentary or chromatolytic degeneration of the nerve cells, suggesting a preferential localization of the pathological process has been observed (Fig. 171).

In all cases subjected to thorough histological study, abnormalities of the



FIGURE 170 Mega esophagus Section through the middle third of the esophagus showing the Auerbach plexus surrounded by chronic inflammatory tissue with fibroblasts new formed vessels and lymphocytes

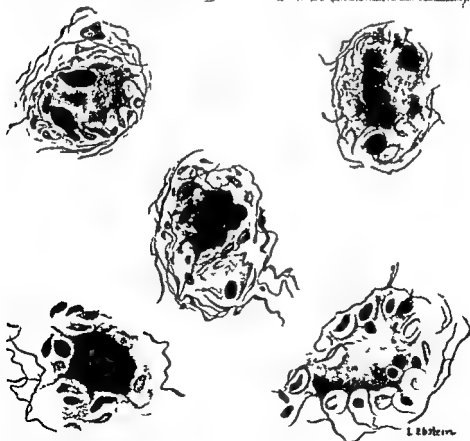


FIGURE 171 Mega-esophagus Middle third of the esophagus Various forms of degeneration of the cells of Auerbach's plexus showing pyknosis chromatolysis disappearance of the nerve fibrils and neuronophagia (E. Etzel.)



FIGURE 174 Roentgen film of a patient with dolicho mega esophagus Type 1. Note the extremely narrow lower segment with enormous proximal dilatation.

picture dominated by the combination of *dysphagia* and *regurgitation* becomes established. Sometimes the early symptoms of the condition are more gastric than esophageal, with epigastric pain soon after eating but without vomiting. Aerophagia may develop. Sometimes there is a spasmodic contraction at the mouth of the esophagus, with intermittent dysphagia limited to this region alone. Finally, in a few the first occurrence may be acute lower esophagospasm with the characteristic dramatic symptomatology (Fig 168, A).

By the time the patient seeks relief, the dysphagia is usually continuous, often with intermittent exacerbations when actual obstruction seems to prevail. Usually it is more pronounced with solids than with liquids, but the reverse may be the case. In fact, it sometimes happens that the only embarrassment is caused by liquids, whereas solids pass without difficulty. The behavior of the esophagus with liquids often depends upon their character and their temperature. For example, plain water may cause no trouble while alcoholic drinks will not go through. Sometimes hot liquids are better tolerated, more often it is the

cold drinks which pass without delay. This *paradoxical dysphagia* is far from constant but is diagnostic when it is encountered.

Sometimes there is a bulimic eagerness for food, no doubt because of the fact that so little enters the stomach. In fact patients who have had relief by surgery sometimes relate that they have never before experienced the sensation of a full stomach. As the meal progresses, an aching discomfort may develop and especially a feeling of fullness and weight in the chest resulting from further distention of the esophagus already dilated with food. Some patients find that by bending forward or backward or by straining (like the Valsalva maneuver) they can force some of the food into the stomach and obtain a degree of relief. In many there is relatively little discomfort, even with the esophagus full of food. In others, however, after an unusually large repast a train of events not unlike those produced by mediastinal compression develops. The feeling of oppression is accompanied by dilatation of the veins of the neck and face which imparts a cyanosed appearance, and an expression of anguish. Respiratory distress may be felt with breathing simulating an asthmatic attack. Relief is obtained only by emptying the esophagus of its contents by regurgitation either spontaneous or induced, whereupon the patient resumes a normal appearance.

Regurgitation is a characteristic symptom of mega esophagus, as mentioned. At first it may occur at almost every meal, but as the esophagus becomes larger and holds more, it may take place no more than once a day or every two or three days depending upon the amount of food consumed. In some instances the patient finds that he cannot lie down because of immediate spilling over of the constantly full esophagus and learns that he must sleep with the head of the bed elevated or even propped up in a chair. The spells of regurgitation are not accompanied by nausea and, when the disease is well developed, they appear without premonitory symptoms.

At first the material regurgitated is relatively small in amount and consists of food recently eaten with no evidence of transformation or fermentation. It contains no free hydrochloric acid as would be the case if it came from the stomach. As the disease becomes well established and the esophagus much enlarged the material is regurgitated in larger quantities and is usually partially decomposed. It tends to be semisolid or liquid in character with a sour or even fetid odor depending upon the length of time it has remained in the esophagus. Sometimes particles of food are enveloped with mucus. In many instances the secretion of mucus is singularly pronounced giving rise at times to regurgitation of mucus alone. Sometimes the regurgitation is *selective*, involving either solids or liquids one without the other. In other instances the regurgitation is *paradoxical* when on the occasion of the ingestion of certain meals the patient may regurgitate not what he has just eaten but the remains of a preceding meal.

Patients usually develop methods of their own to avoid the regurgitation which is often embarrassing as well as unpleasant. Some confine themselves to eating and drinking very lightly, spacing each swallow from the others with an interval of several minutes. Others, as soon as they have eaten a meal drink copious quantities of liquid which by its weight appears to press open the

narrow passage and wash the food through. These patients usually learn just how much liquid is required to bring about the desired result. Some make a series of dry swallows after eating, or take a series of deep breaths. Others bend backward and thrust the abdomen forward in an effort to squeeze the meal through the obstructing segment. These maneuvers may help by producing a sudden increase of pressure within the esophagus. The bending motions may give further assistance by tending to straighten out temporarily the angulated lower segment.

If the effort, whatever it may be, is effective, the patient can discern immediately that the food has moved on into the stomach. He often describes the feeling as a sensation of something falling inside, or as though a door or a valve had been opened. This sensation of something opening up when the material ingested finally succeeds in passing through the cardia is entirely characteristic of this disease. There is immediate relief of any discomfort experienced before the release is felt.

Excessive mucus secretion is observed in some of the patients, due probably to stimulation produced by the continued presence of material in the esophagus.

The general condition of the patient depends upon the severity of the functional disturbance. If it is unrelenting, with regurgitation of almost everything eaten, serious malnutrition may ensue. If the dysphagia is not too severe or if successful treatment is employed, the general condition may be relatively good and the patient may lead an essentially normal existence.

In the pronounced cases when the esophagus remains full much of the time, especially in elderly people, serious difficulties may develop because of aspiration of esophageal contents into the respiratory tract. This tends to occur during sleep and may interfere with the patient's rest. Bronchopneumonia, lung abscess, or chronic bronchiectasis may follow and in some instances are severe enough to be responsible for the death of the patient.

Another important complication of the disease is the effect upon the patient's personality, his social life, and his economic effectiveness. The dysphagia, the tendency to regurgitation, and the peculiar techniques often developed to force the food through the cardia lead to such abnormal eating habits that the patient will refuse to eat away from home or even in the presence of members of his own family. Sometimes the amount of time required to eat interferes with his holding a job. These matters in many patients lead to emotional reactions of fear, worry, or embarrassment which set them apart as peculiar individuals. In fact, the prominence of this aspect of the disease in some instances has led to the widely held belief even among physicians that the cause of the condition is actually an emotional imbalance or, to put it in modern terminology, that achalasia of the esophagus is a psychosomatic disease. Anyone who has had the opportunity to examine the striking anatomical abnormalities characteristically found on exposing the esophagus to view at a surgical operation can hardly agree with this opinion. The occurrence of the disease in a psychiatrist who was certain in his own case that there was no background of psychogenic or emotional abnormality lends interesting confirmation of the probability that the disease does not have a psychogenic origin.

ROENTGEN EXAMINATION In order to insure the success of the examination the esophagus should be emptied not only by the usual regurgitation often induced by the patient, which always leaves a more or less abundant residue behind but also by actual lavage using a large rubber tube. In many instances, when the esophagus is exceptionally large, a grayish mass can be seen occupying the entire mediastinum before any opaque mixture is given. The examination should be started with a few spoonfuls of thick barium followed by the ingestion of a fluid mixture. Large quantities of the latter are required however, in order to obtain an accurate image of the extent of the dilatation. The examination should be made in the erect position behind the fluoroscopic screen.

Films should be obtained as a record of the findings for future comparison. The anteroposterior and right anterior oblique positions provide the best views.

The *fluoroscopic* examination should include successively a study of the manner in which the esophagus fills, the changes in the dynamics of the organ (peristalsis), the functional behavior of the cardia and the function of the stomach and intestine which are much too often neglected.

If the mixture is given in small amounts it will be seen to slide along the walls of the esophagus forming parallel streaks of barium separated by clear zones. The opaque substance often never reaches the cardia but remains attached to the esophageal walls. If given in sufficient quantity the barium meal accumulates above the narrow supracardiac segment outlining the lower portion of the dilated esophagus. If the esophagus has not been emptied before the examination, the barium at first forms a level floating on top of the liquid contents. After a few moments it falls in flocculations like snow through the retained liquid and accumulates at the bottom of the dilated organ where it outlines the more or less irregular and fusiform profile of the lower portion as the latter lies upon the diaphragm (Fig. 175). As more material is ingested the dilated upper

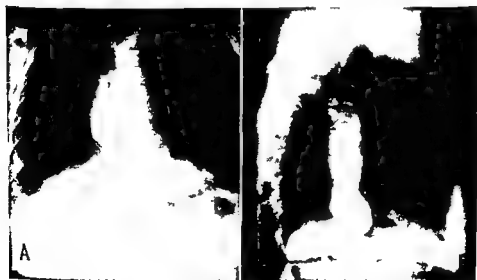


FIGURE 175 A B Roentgen views postero-anterior and lateral of a patient with Type 1 mega-esophagus (Lee McGregor)



FIGURE 176 · Roentgen film of a patient with Type 2 achalasia showing marked delay in the emptying of the stomach characteristic of many cases of achalasia

portion of the esophagus is outlined, the extent depending upon the amount of opaque liquid given

In the Type 1 cases under consideration there is rarely much evidence of peristaltic activity. In fact, as Santy has observed, in this form the esophagus is "asystolic" and inert. It fills impassively like a tub and remains filled for long periods of time without any evidence of peristalsis or tendency to empty itself.

The behavior of the lower segment, which controls the passage of material through the cardia, is contradictory and variable. Sometimes after having filled the dilated esophagus, the barium filters progressively and slowly into the stomach through a narrow aperture which can be seen several minutes after the ingestion of a given quantity of opaque liquid (Fig 174). Sometimes as much as 200 cc. of liquid must be swallowed before this segment can be seen. Sometimes the passage through into the cardia is sudden. This may often be induced by a swallow of water. In some the evacuation takes place in sudden spurts, with intervals of inactivity between.

Often the behavior of the lower segment depends upon the manner of ingestion of the barium and the quantity taken. The passage through the cardia may be normal when the barium is ingested slowly, whereas in the same patient if it is swallowed rapidly a complete blockage results. Sometimes the first few swallows will pass easily, but the remainder of the barium meal is held up often four or five hours at a time the stomach meanwhile having emptied itself completely.

There is then no consistency in the behavior of the epicardial segment with regard to the material swallowed. Esophageal retention is far from being permanent in all patients. There are periods of remission during which the esophagus empties without incident. In fact, some cases of mega-esophagus manifest an essentially normal behavior in this respect.

The physiological behavior of the stomach is always of interest, especially when it is recalled that pylorospasm or at least a functional delay in the emptying of the stomach occurs in some patients with mega-esophagus. Independent lesions such as ulcers or carcinoma must of course be looked for, but one finds often a state of gastroparesis with delayed emptying, as in Figure 176. Sometimes the stomach is small, contracted and hypertonic because of a neuromuscular disturbance which is not confined to the esophagus but involves other portions of the gastrointestinal tract as well. The importance of this observation is too little realized but it has a definite bearing upon the results of treatment and the occurrence of certain complications in patients with achalasia of the esophagus.

Films made with the esophagus full of barium demonstrate various shapes depending upon the degree of dilatation and elongation with the resulting tortuosity.

Type 2 (Approximately 30 per cent of the cases)

A type of achalasia less frequent than that already described is characterized by the fact that the esophagus, though dilated and hypertrophied above the lower segment is never so large as in Type 1. Little tendency to elongation is observed, and the appearance of the lower third is fusiform rather than sigmoidal (Fig. 177). In this group also, although the lower or supracardial segment is narrow in comparison with that of a normal esophagus it is not so small in diameter as in Type 1 cases and is not atrophic in appearance. In fact, in this

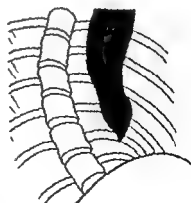


FIGURE 177 Copy of a roentgen film to show diagrammatically the shape of the esophagus in Type 2 achalasia

type there is always a certain degree, often rather pronounced, of hypertrophy of the circular muscle fibers of the narrow lower segment suggesting that spasm of these fibers may be an element of some importance in the evolution of the disease

In keeping with this finding, it is characteristic of the patients with this type of achalasia that they experience, at least in the beginning and sometimes throughout the duration of the disorder, a variable degree of pain characteristic of esophagospasm. This occurs in attacks, usually after eating or during the progress of a meal. It is felt deep within the lower portion of the chest behind the sternum and radiates classically to the interscapular region or the base of the neck following the sensory distribution of the vagus nerves

The volume capacity of the esophagus in this type is never so large as in dolicho-mega-esophagus, and the amount of regurgitation is not so great, though its frequency of occurrence may be much the same

The other manifestations are identical with those of Type 1, including the varied efforts of the patient to force the food down, the dangers of aspiration and the emotional reaction of the patient with its variable social consequences

ROENTGENOLOGICAL EXAMINATION of patients with Type 2 achalasia is



FIGURE 178 Film of Type 2 achalasia showing peristaltic contractions

FIGURE 179 Roentgen film of a child age 11 years with Type 2 achalasia (Now at the age of 22 years the patient is perfectly well operation performed at age 11 years)



characteristic and definitely different from that in Type 1. There is usually the evidence of hyperperistalsis, the waves ending at the cardia without evacuating the esophagus (Fig. 178). These give way to waves of antiperistalsis by which the liquid refluxes violently toward the upper extremity of the organ where it meets a new obstacle in the tightly contracted cricopharyngeus muscle at the mouth of the esophagus. Finally the barium may be expelled by regurgitation through the mouth above or pass little by little into the stomach below. Periods of quiet may then ensue, to be followed by waves of erratic purposeless peristalsis. Figure 179 illustrates the typical appearance of the esophagus in a patient with this type of the disease.

There is no evidence that this form of achalasia represents a phase of development of the disease leading to the striking changes observed in Type 1. In fact, patients with Type 2 achalasia have been observed for as long as nineteen or twenty years without showing any change in the roentgen appearance of the esophagus or experiencing any appreciable change in the symptoms.

Exploration with a Bougie

If attempted exploratory bougienage should be carried out with bougie provided with an olivary tip. A slight resistance may be felt at the mouth of the esophagus but this is usually overcome by gentle traction. The passage of the instrument is then unimpeded until the lower end is reached. At this point the results are variable but the intensity of the contraction and its resistance to pressure vary in a curious fashion depending upon

type there is always a certain degree, often rather pronounced, of hypertrophy of the circular muscle fibers of the narrow lower segment suggesting that spasm of these fibers may be an element of some importance in the evolution of the disease

In keeping with this finding, it is characteristic of the patients with this type of achalasia that they experience, at least in the beginning and sometimes throughout the duration of the disorder, a variable degree of pain characteristic of esophagospasm. This occurs in attacks, usually after eating or during the progress of a meal. It is felt deep within the lower portion of the chest behind the sternum and radiates classically to the interscapular region or the base of the neck following the sensory distribution of the vagus nerves.

The volume capacity of the esophagus in this type is never so large as in dolicho-mega esophagus, and the amount of regurgitation is not so great, though its frequency of occurrence may be much the same.

The other manifestations are identical with those of Type 1, including the varied efforts of the patient to force the food down, the dangers of aspiration and the emotional reaction of the patient with its variable social consequences.

ROENTGENOLOGICAL EXAMINATION of patients with Type 2 achalasia is



FIGURE 178 Film of Type 2 achalasia showing peristaltic contractions

FIGURE 179 Roentgen film of a child age 11 years with Type 2 achalasia (Now at the age of 22 years the patient is perfectly well operation performed at age 11 years)



characteristic and definitely different from that in Type 1. There is usually the evidence of hyperperistalsis, the waves ending at the cardia without evacuating the esophagus (Fig. 178). These give way to waves of antiperistalsis by which the liquid refluxes violently toward the upper extremity of the organ where it meets a new obstacle in the tightly contracted cricopharyngeus muscle at the mouth of the esophagus. Finally the barium may be expelled by regurgitation through the mouth above or pass little by little into the stomach below. Periods of quiet may then ensue, to be followed by waves of erratic purposeless peristalsis. Figure 179 illustrates the typical appearance of the esophagus in a patient with this type of the disease.

There is no evidence that this form of achalasia represents a phase of development of the disease leading to the striking changes observed in Type 1. In fact patients with Type 2 achalasia have been observed for as long as nineteen or twenty years without showing any change in the roentgen appearance of the esophagus or experiencing any appreciable change in the symptoms.

Exploration with a Bougie

If attempted exploratory bougienage should be carried out with a soft bougie provided with an olivary tip. A slight resistance may be encountered at the mouth of the esophagus, but this is usually overcome by gentle pressure. The passage of the instrument is then unimpeded until the lower segment is reached. At this point the results are variable but the intensity of the obstruction and its resistance to pressure vary in a curious fashion depending upon the time

of the investigation and the characteristics and the diameter of the bougie employed. This variability is a sign characteristic of the disease. Sometimes the smallest caliber bougie meets an obstruction although a large size paradoxically will pass without difficulty. In order to verify the presence of an obstruction it is always necessary to ascertain, if need be by fluoroscopy, that the bougie is not curled up in the redundant lower esophagus or that it has not ended up in a blind pouch above the cardia. Force applied in the latter case is sure to result in perforation followed by mediastinitis. In general, this method of examination should not be employed because of the dangers involved in its use and the inadequacy of the information it provides.

Esophagoscopy

The performance of an esophagoscopy often provokes a marked degree of reflex salivation. The tube at first encounters a more or less pronounced degree of spasm at the mouth of the esophagus, which may extend actually into the cervical portion. Once this region has been passed, one is struck by the degree of lateral motion of the esophagoscope which is possible. There seems to be no limit to the amount of movement allowed, but the inclination to test the extent of this must not be indulged because of the danger of injuring the mucous membrane. The esophagus always contains a certain amount of debris and mucus. After evacuation by aspiration or by lavage with a large tube (Fig 57), the mucosa can be examined. It is seen to be heaped up in transverse folds, one superimposed upon another, which fall down in front of the tube as it advances, interfering with the visibility (Fig 180). This appearance is completely characteristic. It varies with the duration and extent of the disease and the presence or absence of esophagitis. When the stasis is complicated by inflammation, the mucosa appears congested with areas of vascular dilatation, sometimes even of hemorrhage, which may lead to the erroneous diagnosis of neoplasm. In a more advanced stage of inflammation the mucosa takes on a grayish tint, with here and there reddish areas of esophagitis alternating with white areas of leucoplakia (Fig 181).

The examination of the supracardial segment is often difficult because of the presence of abundant mucoid secretions which cannot be completely removed by aspiration because of the angulations of the esophagus in the redundant lower portion. Usually the cardiac orifice appears normal, but it is firmly closed and tightly contracted with the resulting innumerable mucosal folds (Fig 182). It appears at the bottom of a long, deep, narrow funnel formed by the constricted lower segment of the organ (Fig 180 also Plate III, 7).

If the examiner attempts to pass the tube through the cardia, a rolled up appearance is produced like the cloaca of a fowl (Fig 183). No force should be exerted. Injection of a local anesthetic at this point usually permits enough relaxation in a few moments to make penetration into the stomach possible. If, however, there should be a secondary cicatricial stenosis, this cannot be accomplished and a filiform bougie may be the only object which can be passed.

In contrast with the cases in which the lower segment is more or less tightly closed as if obstructed, there are innumerable others in which, even with-

out pressure on the esophagoscope, the orifice of the cardia will open and allow the passage of the instrument easily without anesthetization. This lack of resistance of the cardia seems to prove that the disorder is characterized much less often by spasm than by failure of the sphincteric mechanism to relax. In other words, there is a dysectasia. The stimulation provoked by the pressure of the esophagoscope suffices to cause the relaxation and opening of the lower segment, although the arrival of food in the same area does not.

FIGURE 180 Folds of the esophagus in a patient with achalasia and enormous dilatation: endoscopic view.



FIGURE 181 Leucoplakia of the mucosa in a case of mega-esophagus (E. Fitzel.)

of the investigation and the characteristics and the diameter of the bougie employed. This variability is a sign characteristic of the disease. Sometimes the smallest caliber bougie meets an obstruction although a large size paradoxically will pass without difficulty. In order to verify the presence of an obstruction it is always necessary to ascertain, if need be by fluoroscopy, that the bougie is not curled up in the redundant lower esophagus or that it has not ended up in a blind pouch above the cardia. Force applied in the latter case is sure to result in perforation followed by mediastinitis. In general this method of examination should not be employed because of the dangers involved in its use and the inadequacy of the information it affords.

Esophagoscopy

The perforation in esophagoscopy often provokes a marked degree of reflex salivation. At first encounters a more or less pronounced degree of spasm at the entrance of the esophagus, which may extend actually into the cervical portion. After the region has been passed, one is struck by the degree of lateral motion of the esophagoscope which is possible. There seems to be no limit to the amount of movement allowed, but the inclination to test the extent of this motion is indulged because of the danger of injuring the mucous membrane. The esophagus always contains a certain amount of debris and mucus. After evacuation by aspiration or by lavage with a large tube (Fig. 57), the mucosa can be examined. It is seen to be heaped up in transverse folds, one superimposed upon another, which fall down in front of the tube as it advances, interfering with the visibility (Fig. 180). This appearance is completely characteristic. It varies with the duration and extent of the disease and the presence or absence of esophagitis. When the stasis is complicated by inflammation, the mucosa appears congested with areas of vascular dilatation sometimes even of hemorrhage which may lead to the erroneous diagnosis of neoplasm. In a more advanced stage of inflammation the mucosa takes on a grayish tint, with here and there reddish areas of esophagitis alternating with white areas of leucoplakia (Fig. 181).

The examination of the supracardial segment is often difficult because of the presence of abundant mucoid secretions which cannot be completely removed by aspiration because of the angulations of the esophagus in the redundant lower portion. Usually the cardiac orifice appears normal, but it is firmly closed and tightly contracted with the resulting innumerable mucosal folds (Fig. 182). It appears at the bottom of a long, deep, narrow funnel formed by the constricted lower segment of the organ (Fig. 180, also Plate III, 7).

If the examiner attempts to pass the tube through the cardia, a rolled up appearance is produced like the cloaca of a fowl (Fig. 183). No force should be exerted. Injection of a local anesthetic at this point usually permits enough relaxation in a few moments to make penetration into the stomach possible. If, however, there should be a secondary cicatricial stenosis, this cannot be accomplished and a filiform bougie may be the only object which can be passed.

In contrast with the cases in which the lower segment is more or less tightly closed as if obstructed there are innumerable others in which, even with

out pressure on the esophagoscope, the orifice of the cardia will open and allow the passage of the instrument easily without anesthetization. This lack of resistance of the cardia seems to prove that the disorder is characterized much less often by spasm than by failure of the sphincteric mechanism to relax. In other words there is a dysectasia. The stimulation provoked by the pressure of the esophagoscope suffices to cause the relaxation and opening of the lower segment, although the arrival of food in the same area does not.

FIGURE 180. Folds of the esophagus in a patient with achalasia and enormous dilatation. endoscopic view.



FIGURE 181. Leucoplakia of the mucosa in a case of mega-esophagus (E. Etzel).

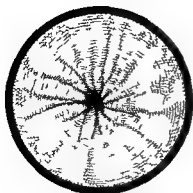


FIGURE 182 Drawing made at esophagoscopy to show the funnel shaped appearance at the lower end in a case of achalasia (After Guisez)



FIGURE 183 Drawing made at esophagoscopy showing hypertonic occlusion of the cardia with a circumferential swelling having jutting lips separated by a narrow fissure (cervix like appearance)

Complications of Mega-Esophagus

Complications arise chiefly in patients who because of either personal neglect or improper supervision go for long periods of time without treatment. They may be local, regional, or generalized. Of the complications occurring locally in the esophagus itself, by far the most frequent is *esophagitis*. The inflammatory changes may be superficial, consisting merely of hyperemia and swelling of the mucosa, or they may be further advanced in the form of granularomatous thickening with an irregular bulging appearance and ease of bleeding which may lead to the suspicion of carcinoma. Finally, if the condition is of long standing, ulcerations may develop with deep seated inflammatory thickening and a tendency to cicatricial contraction. When pronounced the esophagitis adds greatly to the burden of the patient and if neglected the increase of obstruction may be so great that the dysphagia becomes complete. The patient becomes malnourished even to the point of cachexia with peripheral nutritional edema and desquamation of the palms and soles of the feet. Examples of this were observed in France during the last war.

If the ulcerations become deep, serious hemorrhages may occur and perforation is not unknown. Periesophagitis is an almost universal development in any case of severe esophagitis whether in a mega-esophagus or not.

Other complications include the development of *leucoplakic transformation* of the mucosa in the lower portion above the narrow segment, as already mentioned (Fig 181), and of *neoplastic changes*. The latter occurrence raises an interesting question. The development of carcinoma in mega-esophagus usually appears to be primary and not the result of so-called precancerous inflammatory

changes. Whether or not the abnormalities of the mucosa characteristic of leucoplakia represent a precancerous transformation as a step towards the development of epitheliomatous neoplastic growth is not exactly certain, but the possibility must be admitted. If local irritation is an etiologic factor, there is plenty of provocation in patients with achalasia because of chronic stasis and the decomposition of food which produces irritating substances. If the reported statistical occurrence is correct (3 to 8 per cent depending upon the source), the incidence of carcinoma in mega-esophagus is definitely greater than in persons with a normal esophagus. In the author's experience the occurrence is 4 per cent (R. H. S.).

From the *clinical viewpoint*, cancer engrafted upon a case of mega-esophagus progresses to an advanced phase of its development before its discovery. This is because of the fact that the large diameter of the esophagus prevents the development of obstruction early in the evolution of the tumor. Sometimes the carcinoma is discovered first, usually as a result of symptoms arising from local spread into the bronchi or lungs or from distant metastases. Lung abscess, pneumonitis, empyema or esophagobronchial or tracheal fistula may develop before the presence of anything unusual other than the underlying mega-esophagus is suspected (Figs. 184 and 185).

Occasionally a diverticulum of the pulsion or juxtaspincteric type may develop in a mega esophagus. As would be expected, the majority of these are of the epiphrenic variety arising just above the abnormal lower segment in the lower esophagus. In a series of eighteen patients with mega esophagus who also had a diverticulum reported by J. Jacquemin one was pharyngo-esophageal, thirteen were epiphrenic, and four were of the traction type occurring as always

FIGURE 184 Photograph of the specimen removed at operation. Case of carcinoma arising in mega-esophagus with a fistulous communication into the lung where there was a large putrid abscess. Partial esophagectomy and total pneumonectomy (right lung).

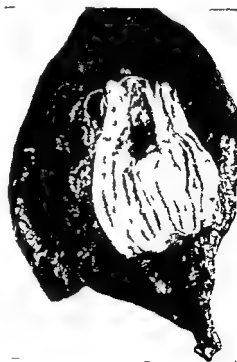




FIGURE 185 Roentgen film of a patient with Type 2 achalasia treated five years previously by esophagomyotomy showing large annular filling defect in the superior mediastinal segment produced by a carcinoma

in relation to adherent lymph nodes in the region of the major bronchi. For a discussion of diverticula of the esophagus see Chapter 11

Among regional complications, other than those which result from the local spread of a carcinoma superimposed upon a case of mega-esophagus, should be mentioned the occurrence of tracheal obstruction due to pressure of the over distended esophagus. This is noticed especially when there is associated cricopharyngeal spasm which prevents the release of intra-esophageal pressure. Sometimes obstruction of the venous return from the head and neck by pressure upon the superior vena cava or hoarseness of the voice from pressure on the recurrent laryngeal nerve is observed. Cardiovascular disturbances including syncopal attacks as a part of disturbances of the cardiac rhythm, notably sinus bradycardia demonstrated by electrocardiography may develop. Finally, pulmonary disorders including pneumonitis, lung abscess, and atelectasis resulting from aspiration of regurgitated esophageal contents are fairly frequent regional complications of the disorder. Collapse of the right lung has been known to be the result of occlusion of the main bronchus on that side from pressure of an unusually distended esophagus of the dolicho mega esophagus type (Type I).

General complications are merely the changes secondary to inanition in advanced and neglected cases. Wooler has called attention to the occurrence of rheumatoid arthritis in patients with severe degrees of achalasia.

Differential Diagnosis

The diagnosis of achalasia of the esophagus presents no serious problem for one who is familiar with the condition and who knows how to carry out an

examination of this organ. Two different situations occur, depending upon the stage of evolution of the disease.

When the affliction is relatively recent and is manifested only by dysphagia, the important error to avoid is the misinterpretation of the nature of this dysphagia, mistaking it for one of the numerous manifestations observed in neurotic or emotional persons. The frequency among neurotic subjects of complaints of stifling, inability to get enough air, the feeling of a ball or bulbous swelling in the throat (*globus hystericus*), or of slight retrosternal twinges of pain with or without extrasystoles of the heart should be kept in mind. Sometimes if there is sharp pain, the condition is mistaken for *angina pectoris*.

A careful analysis of the discomfort or pain with regard to its radiation, its tendency to occur immediately after eating, its association with disturbances of digestion, and a tendency to regurgitation should lead to a suspicion of the esophagus as the cause. No mistake will be made if in every such patient a roentgen examination of the esophagus is performed.

The condition need not be confused with aerophagia, which can be recognized easily by observing the repeated swallowing or gulping motions made by the patient. All patients afflicted with flatulent dyspepsia and wrongly considered air swallows show a voluminous pouch of air in the stomach and esophagus on fluoroscopy. This can scarcely lead to confusion.

The diagnostic problem becomes different when the predominance of dysphagia over the other lesser difficulties begins to call attention directly to the esophagus. The variability of the dysphagia and its intermittent occurrence often are not sufficiently characteristic to exclude the possibility of a cicatricial stenosis or of an obstructing tumor. Actually, these variations in the manner of appearance, the intensity, and the duration of the dysphagia may be encountered in certain cases of organic stenosis complicated by intermittent esophagospasm.

Aortitis, by producing local irritation, can lead to a form of spasm resembling that due to disturbances of the innervation.

Compression of the esophagus by a mediastinal tumor or an aneurysm may be a source of confusion, but the symptoms and particularly the roentgen ray appearance should set one straight immediately.

The chief diagnostic problem is to differentiate the condition from *carcinoma*. The general appearance of the patient does not provide any help. It is among those who are aged, thin, and debilitated, sometimes almost cachectic in appearance, that cancer may be suspected when in reality they are merely the possessors of an infected mega-esophagus. Conversely, one may observe patients afflicted with cancer of the esophagus who maintain a healthy appearance for a long period of time. The distinction cannot be made without the help of the roentgen examination and often of esophagoscopy. In fact, because of the rather frequent coexistence of carcinoma in cases of mega-esophagus, it should be established as an inflexible rule that an esophagoscopy should be performed in every patient with achalasia.

The roentgen appearance of the esophagus in the majority of instances of achalasia, particularly in Type I, is so characteristic that the diagnosis can be made immediately. Rarely there may be enough dilatation proximal to a small constricting carcinoma of the lower esophageal segment to cause confusion with

mega-esophagus of the fusiform or Type 2 variety in spite of every effort made at fluoroscopic examination to differentiate the two conditions. It is in this sort of case particularly that esophagoscopy or cytological studies of washings taken from the esophagus are of so much help.

Esophagoscopy likewise may be the only method to distinguish some cases of cicatricial stenosis due to chronic esophagitis or to chemical burns. Here again it is with the Type 2 cases which show a simple fusiform dilatation that the confusion arises.

In the more advanced cases the clinical picture is dominated by the frequent occurrence of regurgitation from the esophagus, but as mentioned before this need not be confused with vomiting of gastric contents because of the obvious differences in the characteristics of the material ejected. In a few instances of gastric disturbance, however, the vomiting is so prompt that this difference may not be so striking. These are chiefly the vomiting resulting from a distended stomach due to overeating, and the gastric intolerance of certain neurotic subjects or that which occurs in scirrhus carcinoma, acute gastritis, and hourglass stomach.

Roentgen examination of the stomach as well as of the esophagus serves to clear up any doubts as to which may have given rise to the clinical manifestations.

Treatment

Dietary Measures

Many times the patients themselves discover how to improve their condition by modifying their dietary and eating habits. Experience teaches them that a certain regimen suits them better than any other. They avoid rapid eating, which sometimes appears to initiate their difficulty and always aggravates it. They learn the necessity of adequate admixture of the food with saliva, slow, thorough mastication, and slow deglutition of small, well morcellated mouthfuls often washed down each time with a swallow of liquid. Solid foods are usually poorly tolerated. The food should be semisolid or liquid. Soups, purees and finely chopped meats should make up the bulk of the diet. There is no absolute rule, however, as certain patients can accommodate rather heavy or even irritating meals. It is wise to advise against eating just before retiring, in order to put the esophagus at rest during the night.

As already mentioned, these patients often realize that certain maneuvers or gymnastic movements after eating encourage the passage of food through the esophagus. These maneuvers may be suggested to others who may not have tried them. Certain movements which tend to straighten the elongated angulated esophagus may be helpful. This may be done by throwing the head backward like a chicken as it drinks water, or by bending the chest backward and taking a deep breath. The rapid ingestion of a swallow of liquid, taking carbonated drinks making several swallowing motions in succession followed by closure of the glottis and succeeded by straining efforts to increase the intrathoracic pressure, or the gulping of air may provoke an almost immediate emptying. Sometimes a few minutes elapse before the desired result is obtained.

Medication

The administration of parasympatholytic drugs is ineffectual. Belladonna, atropine, Banthine, etc. do not improve the situation; on the contrary, they make it worse. Nevertheless, small doses of atropine are sometimes advised. In normal subjects, atropine may provoke esophagospasm. At fluoroscopy the passage of a swallow of barium through the esophagus is slowed down and actually stopped completely at intervals after the injection of 0.25 to 0.5 mg. of atropine. In fact, atropine makes swallowing slower, more difficult, and less effectual. It increases sphincteric spasm. It should never be administered in achalasia.

The action of sympathomimetic agents like Adrenalin is nil. The same is true of the sympatholytic drugs like ergotamine.

Medicaments like papaverine which relax smooth muscle fibers produce violent antiperistalsis.

Amyl nitrite, however, after several seconds may bring about a rapid and abundant evacuation of the esophagus into the stomach. This serves in some instances to distinguish mega esophagus from dilatation secondary to organic stenosis. Nitroglycerin has been employed but cannot be used with regularity because of the unfavorable side effects.

Acetyl beta-methylcholine (Mechoyl) may be administered in daily doses of 250 mg. diluted with water and given usually at breakfast time. The results appear to be temporary.

Vitamin B₁ given as an injection or tablet or by increasing the intake of green vegetables or by drinking beer for its yeast content may be tried on the theory evolved by the Brazilians that deficiency of this vitamin is an important etiological factor.

It may be worth while, particularly with spasmophilic children who have mega esophagus, to try the effect of the administration of calcium during courses of exposure to ultraviolet rays.

In neurotic subjects rest, outdoor exercise, hydrotherapy, and other measures of a general hygienic nature are useful.

Medicinal therapy alone, however, unless it has some psychological value, leads usually to disappointing results.

Instrumental Dilatation

For the great majority of patients afflicted with mega-esophagus the methodical passage of a bougie through the cardia is the best form of treatment. Soft bougies with olivary tipped, tapering ends may be employed. If certain rules are followed, bougienage usually succeeds in effecting a definitive cure though not always. It will, at least in the majority of instances, bring about a degree of amelioration which is compatible with a normal existence.

The dilatation must be slow and gradual. As in the urethra the sound should act by stretching rather than by divulsion of the tissues. One might ask in the cases in which the cardia is easily entered whether bougienage does not act as a physiotherapeutic agent by bringing about a happy alteration of the disordered neuromuscular mechanism rather than as a means of dilatation.

It is wise not to pass more than two or three bougies at a single sitting and always to begin with a size smaller than that reached at a preceding treatment.

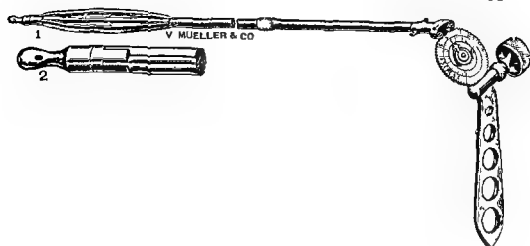


FIGURE 186 Mosher type mechanical dilator (V Mueller & Co)

The bougienage should hardly ever be repeated more than once or twice a week.

In difficult cases in which the bougie cannot be made to penetrate the orifice of the cardia, the esophagoscope should be used in order to make it possible to pass the instrument with visual control. The endoscopic tube by untolding the esophageal mucosa and by making it possible to infiltrate the region of the cardia with a local anesthetic agent greatly facilitates the passage of the bougie.

Another valuable method for entering the cardia under difficult circumstances is the use of three filiform bougies passed together. One will usually pass, thanks to the pushing aside of the mucosa by the others. The filiform bougie, once it has passed, should be left in place for twenty-four hours or more if possible, when it will serve as a guide for a bougie of larger size.

Guisez has pointed out correctly that in order to secure the widest possible dilatation without the discomfort occasioned by compression of the larynx several bougies should be passed at one time. This is based upon the fact that the esophagus lends itself more readily to dilatation in a transverse direction. Once a bougie of small size (20 or 22) has been introduced through the cardia, it is easy to slip in a larger bougie (30 to 35) and sometimes in between these a third still larger (35 to 40) thus producing a forceful dilatation superior to that produced by a single bougie of large caliber (58 to 60) which would press painfully against the cricoid cartilage.

Another useful instrument is the *mercury bougie* introduced by Inurrigo in 1906. This is a bougie made of soft red rubber of variable dimensions containing an amount of mercury commensurate with its size. In starting its use in a given patient it is wise to introduce the mercury bougie under a few centimeters. Carried along by its weight it descends steadily. It is stiff and becomes curled up in the tortuosities of a dilated elongated esophagus. It is flexible enough to slide by the wall. By its constant pressure it easily dilates the wall of the cardia. It should be allowed to remain in place for five or ten minutes. In subsequent dilations it may be used again.

periods depending on the tolerance of the subject. The patient after a certain amount of experience often succeeds in passing the bougie himself. He can then carry out the treatment at home as frequently as required.

The maneuver does not involve any danger to the patient provided the bougie is not introduced too rapidly.

A paradoxical fact is that a large size is usually better tolerated than a small one.

At first the treatments are carried out every few days, but in favorable cases the interval between can be elongated rapidly until they are required only once in several weeks or even months. Finally the instrument may be kept in reserve to be used only when dysphagia reappears.

The majority of authors agree that rapid dilatation by means of mechanical devices such as that developed by Mosher and others is brutal and dangerous.

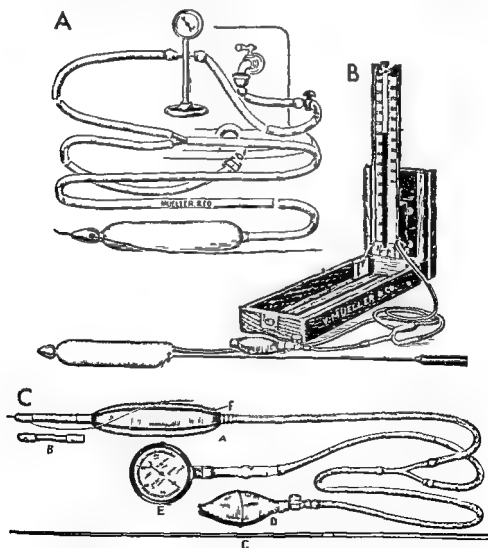


FIGURE 187 Illustrations of three types of dilator used in the treatment of mega-esophagus. A Plummer hydrostatic dilator used on a water faucet. B Sippy dilator for use with any standard mercury manometer. C Mosher air pressure dilator (V Mueller & Co.)

(Fig. 186) In spite of this, various operators have reported remarkable results obtained by the use of the *Starck dilator*, which actually tears the mucosa. Examples of long-standing cure have been observed and no complications have been reported. This dilator is introduced under fluoroscopic observation with the patient lying on his left side. It is manipulated until it lies astride the cardia. The dilatation is quick and carried to the point of tearing the narrowed segment. As familiarity with the instrument is gained, the operator's hand learns to appreciate the degree of resistance which must be overcome. The manipulation produces an acute pain which is of short duration in the majority of instances. Usually a single dilatation suffices to produce a permanent result. Sometimes, however, two or more attempts must be made before the treatment succeeds. This method cannot be employed unless there is a sufficient passageway to allow the correct placing of the instrument.

Another instrument more widely used than the former is the *hydrostatic dilator*. This consists of a bougie or sound provided with a balloon which can be placed in the constricted segment and then filled rapidly with liquid. Results are good (Seventy per cent are cured and an additional 20 per cent relieved as reported by Maingot, 75 per cent cured by Allison.) Various modifications of this apparatus have been made and used with satisfaction by many operators (Fig. 187).

In general, the results of treatment by dilatation are satisfactory if not entirely successful. In a few a single treatment produces permanent or at least long-lasting relief from dysphagia. In some it remains necessary to repeat the treatment from time to time, but patients often accept the bondage of dependence upon the use of a bougie and lead fairly comfortable lives. With others, however, the results may be so temporary and ineffectual that something else is obviously necessary to secure the relief desired. Sometimes, because of pain or bouts of fever occurring after the treatment, it is necessary to abandon further attempts. Finally, there are a few patients who cannot tolerate the bougie and who seek surgical relief rather than to submit to further treatment by dilatation.

Surgical Methods in the Treatment of Achalasia

Many operations based upon theoretical considerations have been tried and abandoned. These include left phrenicotomy, cervicothoracic sympathectomy, splanchnicectomy, and resection of the periarterial ganglionic plexus around the left gastric artery. All of these methods of indirect attack have been ineffectual.

Several procedures directed toward altering the anatomical abnormality of the lower esophageal segment have been given up. Among these are division of the cardia performed by forceful trans-gastric finger dilatation analogous to the dilatation of the anal sphincter in the treatment of fissure ani, and efforts to pull the lower esophagus down into the abdomen in the hope that straightening the angulated lower segment will bring relief. The former is too brutal and is likely to be complicated by esophagitis engrafted upon the infection of torn mucosa, and finally by stenosis, with worse dysphagia than before.

Certain procedures ordinarily grouped together under the heading of 'cardioplasty' have been tried and abandoned. These include the *Heyretzki*

procedure which is analogous to the Finney type pyloroplasty, and the *Grondahl procedure* which involves the performance of a *lateral anastomosis* between the fundus of the stomach and the tortuous lower esophagus proximal to the constricted epicardial segment. In the hands of the majority of surgeons who have tried them, these sidetracking procedures, though usually successful at first in overcoming the obstruction, have ended in a high percentage of failures due to esophagitis with resulting cicatrization leading to stenosis and recurrence of dysphagia.

ESOPHAGOMYOTOMY. Although the results reported from various centers are not uniformly satisfactory, the operation which has gained the greatest favor among surgeons throughout the world is that of esophagomyotomy first described by Heller in 1914. This method is pre-eminently successful in the Type 2 cases in which the circular muscle fibers of the lower esophageal segment are hypertrophied. The operation may be carried out either through a high left paramedian abdominal incision or through a lower left thoracotomy incision in the eighth intercostal space.

The *abdominal approach* has the advantage that it avoids opening the chest and that it is less painful than a thoracotomy. It is particularly suited to thin or emaciated patients, especially if the patient is placed in a lordotic position. Its disadvantages are that the exposure of the lower esophageal segment involves carrying out a considerable degree of local dissection and that in general the exposure, particularly of the upper end of the narrow segment, is rather difficult to obtain.

Once the incision is made the left lobe of the liver is reflected by dividing the triangular ligament. This makes it possible to gain access to the peritoneum which must be incised to expose the abdominal portion of the esophagus (Fig 188). Dissection of the periesophageal fascial investment often involves the severing of some of the veins and arteries which are a part of the anastomotic vascular channels in this region (Fig 189). A piece of Penrose drain or of tape is passed beneath the esophagus and used as a means of gradually drawing down the abnormal segment which must be exposed to view throughout its entire length (Fig 190). With a sharp knife a longitudinal incision is then made cautiously through the muscular layers as far as the submucosa. As the last of the circular fibers are severed the underlying mucosa bulges through the cleft which has been made. In order to secure a good result the incision must extend from the cardia just below a point on the thickened dilated portion to a level definitely above the narrowed segment (Fig 191). The length of the incision varies from 4 or 5 to 8 or 10 cm, depending upon the longitudinal extent of the constricted segment. Caution must be exerted as the knife crosses the cardia onto the stomach where the muscularis is thin by comparison and where it is easy to penetrate the mucosa. If this should happen one or two sutures of fine silk are used to close the opening. In this respect the procedure resembles the Fredet-Ramstedt operation for pyloric stenosis.

The *thoracic approach* through a left intercostal incision provides much easier access to the lower esophagus than the abdominal exposure. As soon as the overlying mediastinal pleura is incised the abnormal lower esophageal segment is available without the necessity for any degree of dissection which in

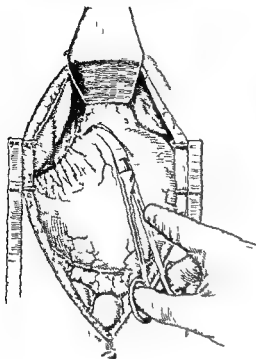


FIGURE 188

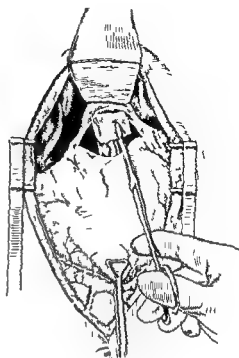


FIGURE 189

FIGURE 188 Heller esophagomyotomy abdominal approach First step exposure of the lower esophagus by incising the peritoneal reflection (After R Soupault)

FIGURE 189 Heller esophagomyotomy dissection of the esophagus with gauze tampon (After R Soupault)

some instances, where an abdominal incision is used, is actually rather considerable. Sometimes the diaphragm at the hiatus must be retracted downward and the esophagus pulled up somewhat in order to gain access to the cardia. The incision through the muscular coat of the involved segment is made exactly as when the abdominal exposure is used but with greater ease. In general this approach is preferable to the abdominal.

Aftercare The recovery period is approximately the same no matter which incision is used. The patient is allowed out of bed early, usually on the second postoperative day. Oral administration of liquids is begun with 30 cc of water each hour on the day after operation, increasing gradually in quantity from day to day until by the fourth or fifth day 120 cc of nourishing liquids may be given every two hours, with water as desired cautiously in between. Soft solids may be started gradually by the fifth or sixth day and by the tenth the patient can be permitted a diet of six small meals consisting of soft solids, chopped meats, and nourishing liquids. Thus he is instructed to use during the first few weeks after discharge from the hospital. If a chest catheter has been used, it may be withdrawn after forty-eight hours.

In every other respect the aftercare is essentially the same as for patients who have had other surgical procedures, depending upon the type of approach used.

Results The relief is immediate and dramatic. The patient can usually recognize the disappearance of the dysphagia with the first swallow of water. As more food is allowed, the joy of eating without hindrance and without effort

becomes a new experience for those who have been troubled all their lives. Some will say that they have never before known how it feels to have a full stomach. As would be expected, the gain in weight is rapid and the freedom from embarrassment and frustration has a beneficial effect upon the personality in patients who have long been considered neurotics.

The long term follow up of patients who have been treated by a Heller operation, however, reveals that the end results are not uniformly excellent. Reports differ from one series to another, but there is a fair degree of uniformity. A summary of experiences submitted by eleven different surgeons in the literature of the past fifteen years reveals that of 233 patients, 153 had excellent results, often spoken of as a cure, fifty were said to be improved, with a good or fairly good result, and twenty-five were unimproved. There were five operative deaths in the entire series. Thus in the usual experience the operation of esophagomyotomy may be expected to cure the patient of his dysphagia without unpleasant or crippling complicating effects in approximately 65 per cent of the cases. It is interesting that in the experience of several different surgeons a repetition of the procedure after an initial failure has been uniformly unsuccessful.

In the author's experience, however, the results have been uniformly excellent (R H S). This disparity, if not due to chance occurrence because of the

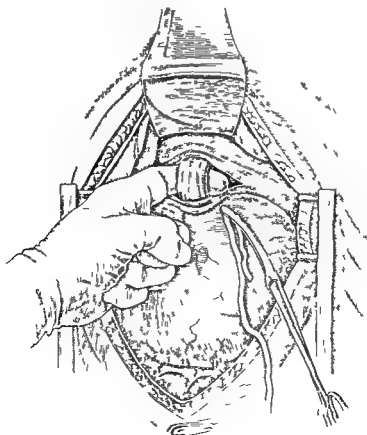


FIGURE 190 Heller esophagomyotomy. Passage of traction tape around the esophagus (J Perrotin).

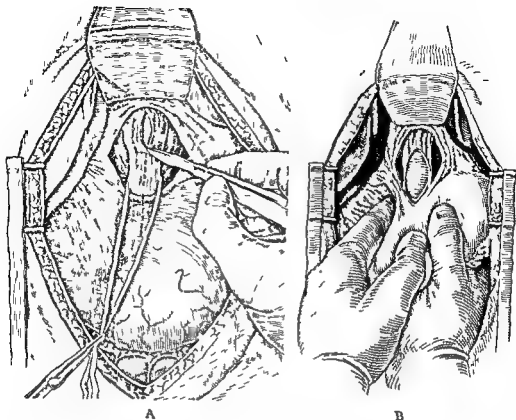


FIGURE 191 Heller esophagomyotomy Line of incision through the muscular layers (A) and completed incision (B) (A After J Perrotin)

smaller number of patients, may be in part at least the result of a special policy with regard to the choice of patients whereby the Heller procedure has been employed only in the treatment of patients with Type 2 achalasia in which the circular muscle fibers of the abnormal segment are actually thickened and hypertrophied

It should always be kept in mind, furthermore, that a certain number of patients with achalasia have a functional disturbance at the pylorus as well which may in part be responsible for some of the unsatisfactory results reported by others (see below)

ESOPHAGOPLASTY BY LONGITUDINAL INCISION AND CIRCUMFERENTIAL CLOSURE In many cases of Type 1 achalasia, where the abnormal lower segment is exceptionally narrow in diameter with atrophic, poorly developed muscular layers, it is preferable to enlarge the narrow portion by the simple maneuver of lengthwise incision with closure of the opening in the opposite direction. This operation was performed first by Wendel in 1909. The operation is performed through the left pleural cavity, using an incision in the eighth intercostal space. The incision of the esophageal wall is made through all layers, starting at but not below the cardia and extending to a point above the narrow segment where the lumen begins to be approximately the diameter of a normal esophagus or a little larger (Fig. 192). Starting at a point midway between the ends of this incision the opening is closed circumferentially, using interrupted sutures of 5-0 silk according to the technique employed for making an anasto-

mosis (mucosal approximation edge to edge, muscular layer edge to edge, and an outer layer to invert) If the incision has been made correctly, the narrow segment is completely eliminated and the lumen where it was is given a diameter approximately that of the cardia, which is always of normal dimensions The operation is not a cardioplasty but is confined to the narrow segment of esophagus alone (Fig. 193)

Care should be exerted to avoid as much as possible any interference with the vagus nerve fibers, the fascial structures about the cardia, and the esophageal hiatus itself At the completion of the procedure the cardia should not be allowed to rise above the margin of the hiatus If the latter is too large or if there is actually a small hiatus hernia, as is sometimes the case, one or more sutures of heavy silk should be placed across the hiatus behind the esophagus to narrow its diameter to the correct dimension These measures assist in the prevention of an abnormal degree of regurgitation from the esophagus into the stomach

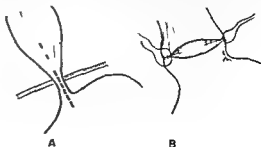
Aftercare The care of the patient postoperatively is essentially the same as that following an esophagectomy except that oral feeding can be advanced at a somewhat more accelerated rate The mortality is relatively low

Results As with esophagomyotomy, the relief from dysphagia is immediate and complete After a safe interval the patients may adopt a normal dietary regimen and the improvement in their condition is rapid In the majority of instances the long-term results are excellent as well On the other hand, if the operation is incorrectly done so that excessive regurgitation from the esophagus takes place or if there should be functional delay in gastric emptying because of pylorospasm or cicatricial stenosis of the pylorus or duodenum esophagitis may develop This if uncontrolled may become severe to the extent of ulceration with the attendant complications of bleeding or cicatricial stenosis

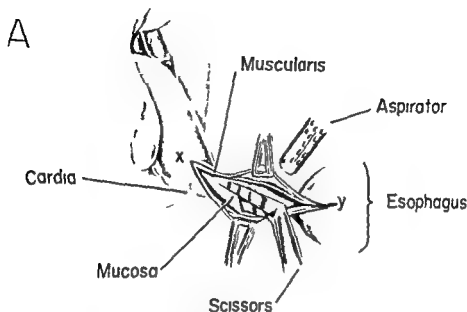
If the patient is one who has difficulty in emptying his stomach he may complain that his dysphagia is unrelieved because the continuously full stomach results in a backing up of food into the esophagus Immediate relief is experienced after the performance of pyloroplasty

It is in these patients also, that chronic anemia may be observed, a condition which is often erroneously attributed to esophagitis whereas the most frequent source is gastritis As an example in a series of eight patients who showed signs of upper gastrointestinal blood loss after esophagoplasty, esophagitis was the source in only two but severe gastritis was found in the other six Gastric ulceration may develop These observations may be explained by the recent work of Dragstedt who showed that the presence of food in the antrum of the stomach causes a great increase in the hormonal stimulation of acid and

FIGURE 192 Diagram showing the principle of esophagoplasty to enlarge the narrow lower segment A Line of incision from wide diameter above to wide diameter below B circumferential closure



Longitudinal Incision



Circumferential Closure

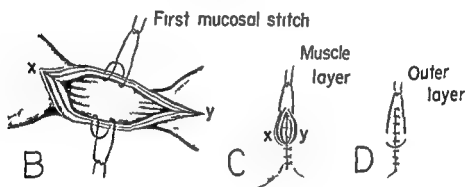


FIGURE 193 Esophagoplasty technique *A* Completion of the incision through all layers
B start of the closure *C D* completion of closure

pepsin secretion which in turn tends to cause or at least to accentuate the gastritis and ulcerations. Here again, relief may be obtained by pyloroplasty, although in intractable cases a partial or even total gastrectomy may become necessary.

The accompanying table summarizes the results obtained in a series of 20 patients treated by esophagoplasty. It should be pointed out that this series includes a number of patients who were operated upon before the importance of avoiding interference with the normal anatomical relations between the gastric cardia and the esophageal hiatus as well as the importance of eliminating any tendency toward stagnation of material in the stomach because of pylorospasm were realized.

Good to excellent relief of dysphagia	18
Esophagitis	3
Stricture	2
Chronic blood loss anemia	5
due to esophagitis	1
due to gastritis	4

In patients operated upon more recently the end results can be expected to be much more favorable

RESECTION of the abnormal segment followed by an esophagogastric anastomosis is a reliable method of overcoming the dysphagia, but it is unnecessarily radical and can be expected to carry a somewhat higher mortality than the procedures just described. The end results are approximately the same as after esophagoplasty, and for the same reasons

Summary

Efforts should be made at first to improve the patient's condition by correcting faulty dietary or hygienic habits so far as his living conditions are concerned. Medicinal treatment may be used as an adjuvant. Failing in these, dilatation by one or the other of the methods described will usually either cure the condition or at least make the life of the patient tolerable. When the condition fails to respond to any of these measures or when the patient is uncooperative resort may be had to surgery. Of the procedures available the Heller operation should be employed wherever applicable. In those cases in which the results from esophagomyotomy may be assumed to be unfavorable, an esophagoplasty by longitudinal incision and circumferential closure should be performed. Surgical resection should be reserved for the rare instances in which either of the other two methods has failed.

Atony and Paralysis

AS A RESULT of the systematic employment of radiology for the study of the function of the gastrointestinal tract and since the development of the esophagoscope, it has been recognized that certain functional difficulties must be attributed to atony or paralysis of the esophagus. Paralysis may be classified under two general categories according to whether it is of *central* or *peripheral* origin.

1 *Esophageal paralysis of central origin* may result from lesions located in either the cortical or the bulbar areas. These may be areas of softening from arteriosclerotic changes, gummas, hemorrhages, traumatism, tumors, the glossolabiopharyngeal syndrome, bulbar syringomyelia, poliomyelitis (bulbar form), or epidemic encephalitis. This type of paralysis of the esophagus is often unrecognized in the presence of other manifestations which overshadow it or direct attention away from its presence. It is indicative, however, of the severity of the nervous system disorder. It is often a premonitory sign of impending bulbar palsy with its inevitably grave prognosis.

In spite of this, some cases of esophageal paralysis of central origin tend to regress with the passage of time if the patient is fortunate enough to survive the acute episode.

2 *Esophageal paralysis of peripheral origin* may result from disorders of the vagus nerves or of the intrinsic innervation of the esophagus itself. Among the latter are the paralysis resulting from infectious diseases like diphtheria, typhoid fever, or botulism, and that due to poisoning such as alcohol, lead, war gases, and the toxemia of pregnancy.

Esophageal paralyses of this sort, the etiology of which is not always clear, often go undiscovered because of the uncertainties of their manifestations and particularly because of their association with disorders of the pharynx, the larynx, or the lungs which mask the true origin of the dysphagia.

Clinical Characteristics

The clinical signs are well known. The patient acts as though he had an obstacle localized rather high in the esophagus. Attempts to swallow give rise immediately to bouts of coughing followed by regurgitation of the food through the mouth and nose. These manifestations are common to cases of esophagospasm as well. Dysphagia of paralytic origin, however, possesses two characteristics which are peculiar to it alone. The *first* is that it is noticed principally on attempting to swallow solid foods, whereas liquids go down freely. This peculiarity, however, exists only if there is no paralysis of the pharynx.

Holzkecht offers the following explanation of this characteristic of the dysphagia in atony of the esophagus. The ingestion of liquids, as has been demonstrated by the physiologists, does not require any peristaltic activity within the esophagus. They may be propelled forcefully from the pharynx and into the stomach through an immobile esophagus. This is why esophageal atony has no effect upon their deglutition. On the contrary, foods of a semisolid or pasty consistency are swallowed with difficulty because they can move along the esophagus only as a result of peristaltic movements which in this condition are lacking. As far as solid foods are concerned they often pass in a normal fashion, at least when the esophagus is atonic but not completely paralyzed. This is explained by the fact that the esophageal musculature although incapable of contracting fully, can still narrow its lumen sufficiently for its walls to come in contact with a large firm bolus and exert upon it a sufficient pressure to make it progress.

Actually there is no uniformity of behavior with regard to the physical properties of the food and it is not unusual to observe instances in which little or no difficulty is experienced with any type.

The *second* characteristic of the dysphagia of esophageal atony is its tendency to grow worse. In the initial stage the esophagus is still capable of reacting under the influence of strong repeated stimuli. There may even be a response to numerous and powerful swallowing efforts on the part of the patient with the mouth empty, showing that reflex peristalsis may not be completely abolished. Certain patients can swallow a bolus of food only after making two or three successive motions of deglutition to start it.

In a more advanced stage with actual paralysis the inability to swallow solids becomes complete. It is then that new difficulties may appear as a result of secondary esophagitis, dilatation of the organ or superimposed spasm. Thereafter the clinical picture loses what little individuality it may have shown before and the signs of paralysis are masked by those of the complications.

There are three other signs, however, which should give rise to the suspicion that the case is one of paralysis. The first of these is the characteristic loss of sensation of the mucosal layer. This is noticed by a failure of the patient to detect the difference between liquids of various temperatures or, even better, at esophagoscopy by the loss of defense reflexes on the passage of the tube. Another is the existence of certain noises noticed by the patient during the spilling of the food into the atonic esophagus and from the esophagus into the stomach. A third is the accumulation of frothy saliva in the pyriform sinuses.

Roentgen Examination

The difficulty of recognizing esophageal atony or paralysis by clinical signs alone is often equaled by the results of roentgen examination which themselves are often open to question. Certain characteristics of the condition, however, are recognizable.

Under the fluoroscope the barium mixture passes through the mouth of the esophagus with difficulty and descends slowly into an inert esophagus which fills completely without any evidence of either peristalsis or antiperistalsis. The swallow of barium finally arrives at the stomach and passes through the cardia without arrest. Twenty minutes later the barium can still be seen occupying the mucosal folds. Sometimes the barium finds its way into the trachea and the valliculae and pyriform sinuses remain filled. The behavior of the three parts of the esophagus will be considered separately.

Mouth of the Esophagus and Pharynx

Difficulty in getting the barium to pass by the mouth of the esophagus in cases of this sort is actually the result of an associated paralysis of the pharynx. This is usually encountered in lesions of the higher centers, as in cerebrovascular accidents or the effects of diphtheria or botulism or in the course of peripheral lesions of the ninth and eleventh cranial nerves.

The roentgenological evidence characteristic of this sort of paralysis is stasis in the valliculae and the pyriform sinuses (Fig. 194), which is best appreciated with only a small amount of barium present. If the paralysis is unilateral the stasis is confined to that side. If bilateral the shadow presents a characteristic appearance like that of a butterfly with outstretched wings (Fig. 194). As another swallow of barium is taken the difficulty with deglutition increases because of the failure of the larynx to close, which permits a certain amount of the material to enter the trachea. This gives the impression of an esophago-tracheal or esophagobronchial fistula (false perforation) (Figs. 195 and 196). It is important to recognize this occurrence in paralysis of the glottis because a recurrent nerve palsy resulting from invasion by a carcinoma of the esophagus may lead one to make an erroneous diagnosis of a fistula when the trouble is due actually to the pharyngeal paralysis.

The opaque material remains in the pyriform sinuses and is not swept on by muscular contraction. In cases of this sort it is possible to differentiate myasthenia gravis from bulbar paralysis by the administration of Prostigmine, which abolishes the phenomenon in the former while in the latter the stasis persists.

The muscular or neural lesion is manifested clinically by the presence of dysphagia which, whether its origin is organic or functional, always shows an underlying deficiency of the normal mobility of the muscles of deglutition. Even a partial inactivity of any one of these parts suffices to disturb the swallowing act, which depends upon this activity and the high degree of coordination of its various constituents.

The lesion may affect either the trunk of the vagus nerve or its principal branch, the recurrent nerve which innervates the mouth of the esophagus.

FIGURE 194 Film showing stasis in the valleculae and pyriform sinuses. Note the presence of barium in the trachea simulating a fistulous communication (The ventricles of Morgagni are clearly defined)



FIGURE 195 Roentgen film showing tracheal aspiration of barium simulating fistula between esophagus and trachea. A Slightly oblique projection B frontal projection. Patient with paralysis of the glottis



FIGURE 196 Film showing barium in the trachea simulating fistula Diverticulum with muscle incoordination leading to stasis in the valleculae and pyriform sinuses with paralysis of the glottis and spilling of barium into the trachea

With this in mind, it is easy to understand why the vallecula sign is encountered so frequently with disorders which are far removed from the mouth of the esophagus, whether they act directly on the intrinsic nerves of the esophagus itself or by way of the main vagal trunks in the mediastinum or the nuclei from which these nerves arise

The objection is often raised that no other sign of vagus nerve involvement may be found, but an affection of the nerve itself is not absolutely necessary to produce the vallecula sign. Movement of the hyoid bone is a necessary accompaniment of normal deglutition and is coupled with that of the larynx and oropharynx. Inaction of any of these parts, as noted before, suffices to disturb the act of deglutition which is so dependent upon the active coordination of all. The dysphagia which accompanies a new growth of the hypopharynx, the esophagus, or the cardia is not, therefore, necessarily due to a stenosis or to stasis above a stenosis, as is commonly assumed but rather to an incoordination in deglutition. In this situation the vallecula sign is helpful because it points to an organic lesion as opposed to a functional variety of dysphagia.

As the pharynx and upper esophagus fill with barium, the opaque column does not have the normal racquet shape. Instead, it appears tubular without any narrowing at the mouth of the esophagus because of the atony of the cricopharyngeus muscle.

Body of the Esophagus

The behavior of a completely atonic esophagus under the fluoroscope varies somewhat depending upon the physical properties of the material ingested. The deglutition of liquids is on the whole not very different from normal except that a certain slowness of passage through the esophagus is observed. The

lumen appears full for a rather prolonged period of time, but it ends up by emptying spontaneously

The thick paste of barium descends very slowly and does not assume the form of a cylinder the length of one's finger as in the normal state. It appears rather as a thin band which extends almost without interruption throughout the entire length of the esophagus and remains there for several minutes, after which it passes little by little into the stomach (Fig. 197). The mixture leaves a residue all along the esophagus as a more or less continuous trail with poorly outlined and ill defined contours.

Several swallows of water suffice to sweep these remains of barium along into the stomach. No peristaltic movement is perceptible.

The functional abnormality is usually accompanied by a slight fusiform dilatation of the organ which is never very pronounced and is noted chiefly in the lower two-thirds. The contour of the esophagus is rather spindle-shaped and its walls appear to be passively depressed by the weight of its contents. This paralytic distention provides a contrast with the appearance of a narrow homogeneous shadow several centimeters in length which appears below it. The latter corresponds to the cardia which, while remaining open, terminates the inferior pole of the shadow of the dilated portion by a more or less thinned out prolongation. This appearance is sufficiently characteristic to avoid confusion with an organic lesion of the cardia or a stenosed appearance due to spasm. Furthermore, unlike the appearance in esophagospasm, there is no arrest of the ingested material in the lower end of the esophagus. The transit is not held up. It merely takes place more slowly than in the normal.

The deglutition of solid material in paralysis of the esophagus gives rise to a characteristic picture. A capsule of barium, for example, progresses by stages with many stoppings and startings. Each of these stops is more or less prolonged, varying from several seconds to a minute or more. Sometimes the capsule moves

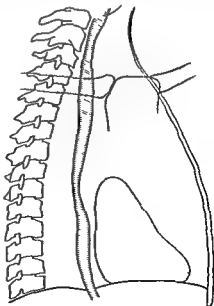


FIGURE 197. Drawing copied from a roentgen film showing the appearance of a paralyzed esophagus (10 minutes after the ingestion of barium: absent peristalsis and failure to empty).

of itself after several seconds, sometimes its further descent is initiated only by a swallow of water or saliva

These arrests of the capsule differ from those observed accidentally in the course of normal deglutition both in their frequency and also in their tenacity which often requires three or four successive swallows of water to overcome. Furthermore, they do not have the permanence of the arrest due to a stenosis and they bear no relation to antiperistaltic movements. The esophageal wall remains inert, relaxed, and immobile.

Röntgen films of themselves are not of much value in the demonstration of paralysis of the esophagus. They show merely a slight fusiform dilatation which without the benefit of fluoroscopic observation has no characteristic appearance.



FIGURE 198. Film showing widening of the lower esophagus and cardia in the condition known as chalasia.

Cardia

Atony of the cardia is characterized by incontinence which permits the reflux of material from the stomach into the esophagus. This is seen, for example, during vomiting and regurgitation. This incontinence is often secondary to lesions located at a distance from the esophagus in the stomach, the duodenum, the gallbladder, or the peritoneum.

It should be recalled that in the newborn infant this sphincter mechanism is often inadequate and that gastro-esophageal reflux is a normal occurrence. Neuhauser and Berenberg, however, have pointed out that if this condition persists or becomes accentuated it can be the cause of vomiting in certain children. This cardio-esophageal relaxation is what they call *chalasia* (Fig 198). In effect, it is the exact opposite of *achalasia* as described by Hurst.

A certain amount of incontinence of the cardia may be observed among the

elderly. During senescence the esophageal hiatus of the diaphragm undergoes atrophic changes, the esophageal walls become atonic, and the normal tonically contracted lower segment becomes enlarged. Often the diameter of this portion in elderly persons may be as large as 4 cm. during complete filling, and even then it is traversed by large stretched out mucosal folds representing exaggerations of the normal. This phenomenon is a part of the picture of aging and explains, according to Harrington, the ease with which eructation can occur in the aged.

Other Examinations

EXPLORATION WITH A BOUGIE reveals complete permeability and insensitiveness of the esophagus and no matter what size is used the instrument can be thrust all the way into the stomach with ease.

ESOPHAGOSCOPY The esophagoscope descends without encountering the least obstacle, in fact much more easily than when anesthesia of the mucous membrane is depended upon to suppress all the reflexes. It passes the mouth of the esophagus without difficulty. The esophageal walls are flaccid, pliable and

FIGURE 199 Drawing made at esophagoscopy to show the endoscopic appearance of the esophagus when paralyzed.



covered with morsels of food. This last finding is inconstant depending upon food intake but when it exists it has great diagnostic value. As the tube progresses it will be noticed that the thoracic esophagus has lost its tonicity. The esophageal walls tend to be flattened one against the other. They may be heaped up in folds like the mucosa in diffuse dilatation. If the patient is recumbent, one gets the impression that the anterior wall falls against the posterior (Fig 199). Finally, the cardia does not offer any more resistance than the cricopharyngeal narrowing.

Prognosis

The number of published reports is so limited that it is not possible to give a definitive answer to the query as to what the future holds for these patients. Much depends upon the underlying cause of the difficulty. Some have apparently recovered completely after a more or less prolonged period of time. This is particularly so with paralysis from botulism or diphtheria. Others have continued to experience difficulty with swallowing compatible nonetheless with the maintenance of a fairly good state of health. Certain others, obliged to reduce their food intake to a notable extent and grown weak and emaciated have maintained a precarious existence thanks to feeding by gavage. An unhappy

existence can be provided by the performance of a gastrostomy, but this may not always be a kindness

Pulmonary complications such as pneumonitis, lung abscess and chronic bronchiectasis are almost sure to develop in patients with pharyngeal paralysis and, in spite of all efforts to alleviate the situation, are frequent causes of death. Occasionally the underlying difficulty may spread to involve the innervation of respiration, thereby bringing about a fatal ending, from respiratory paralysis, as Chevalier Jackson has pointed out

One interesting question is whether or not esophageal atony, when it persists for a long time, can result in so-called idiopathic dilatation. The possibility must be admitted, and in the minds of certain authors (Zenker, Rosenheim, Huber, etc.) atony is considered to be an essential factor in distention. It may be true that atony can lead to dilatation, but this development is far from inevitable

Special Considerations

Diphtheritic Paralysis

The clinician has known for a long time that diphtheria may be complicated by paralysis of the musculature of the pharynx, the soft palate, and the larynx, whether or not it is associated with ocular palsy, with failure of accommodation and polyneuritis. More recently through the employment of roentgen examination it has been recognized that the disease may give rise to esophageal or esophagogastric paralysis as well, with the characteristic evidences described above.

The question arises as to what part should be ascribed to paralysis of the pharynx and what to paralysis of the esophagus in the genesis of the disorder of deglutition. Collet, struck by the accumulation of saliva and ingested liquids in the pyriform sinuses which Chevalier Jackson has taught is a certain sign of esophageal stenosis, does not hesitate to ascribe a great deal of importance to the esophageal factor, but points out that the same sign may result from inertia of the muscular apparatus of the pharynx.

It is now well established, as a result of observations by numerous investigators, that paralysis of the esophagus is a frequent accompaniment of the more easily recognized manifestations of the paralysis observed in this disease. The pathogenesis of diphtheritic paralysis in general remains poorly understood. The same is therefore true when the paralysis involves the esophagus. Various aspects must be considered including the loss of sensation which is always present, as well as inactivation of the intrinsic autonomic innervation and the vagal nuclei as well as the nerve trunks themselves. Although the locus of the effect upon the innervation is not exactly clear, it is obvious that the entire functional activity, both motor and sensory, of the vagus nerves is interrupted by the toxin of the disease.

CLINICAL CHARACTERISTICS. Diphtheritic paralysis of the esophagus appears only among patients who have paralysis of the soft palate as well. Given a patient who is seriously ill and unable to drink without regurgitation through the nose, a physician who is unfamiliar with this association may not think of the involvement of the esophagus and may not recognize the functional difficulties or even the mechanical dysphagia which results. Liquids, though they come

back in part through the nose, will usually pass through the mouth of the esophagus as well, but mouthfuls of food go through only with the greatest difficulty. If the bolus of food after a great effort on the part of the patient can be forced through the cricopharyngeal sphincter, its progress down the esophagus is slow and associated with a sensation of suffocation which is often very distressing. This is accompanied by substernal distress. In the sitting position the food may be washed down with liquids, but the patient can swallow nothing while recumbent.

The swallowing difficulty is greatly aggravated by the presence of spasm which may bring about the paradoxical situation of causing localized spasm in a paralyzed esophagus. Feeding through a Levin tube may become necessary, but if the paralysis involves the stomach as well as the esophagus the situation is grave indeed.

Objective evidence of diphtheritic paralysis of the esophagus includes the curtain sign. If the involvement of the constrictor of the pharynx is unilateral, and accumulation of saliva in the pyriform sinuses indicating paresis of the cricopharyngeus muscle. The latter is of great diagnostic value, but it may occur late in the disease. Hypesthesia or even anesthesia of all the areas in the mucosa from which reflexes normally arise in the pharynx or larynx may be obvious but with the exception of the severest cases this sign is hard to elicit.

THE ROENTGEN EXAMINATION, carried out with the patient standing or at least sitting erect, provides important evidence. As with paralysis from other causes liquid barium descends rapidly, as if falling freely through the mediastinum. A thick paste however, remains in the pharynx and stagnates above the cricopharyngeal sphincter without being able to pass by it. If the mixture, sometimes aided by the introduction of a bougie, succeeds in overcoming this obstacle, it descends slowly through an astyolic esophagus without evidence either of peristalsis or antiperistalsis. It stops at the broncho-aortic constriction, stagnates at the level of the esophageal hiatus and finally passes on through the cardia. The walls of the esophagus however, remain impregnated with little accumulations of opaque substance. This often lasts a long time.

In addition inspection of the pharyngo-esophageal segment discloses the accumulation of material in the pyriform sinuses in the form of spots which look like grape seeds. The typical butterfly shadow cast by these recesses is seen. There is also spilling over into the trachea with release of the cough reflex, ejection of the material toward the glottis and the up-and-down movement of the bulk of the barium as these motions take place.

ESOPHAGOSCOPY is ill advised in these patients.

Confirmation of the diagnosis by finding the Loeffler bacillus in cultures from the throat in addition to the other manifestations of the disease is of course essential.

PROGNOSIS. Diphtheritic paralysis of the esophagus may last as long as two or three weeks sometimes as long as three or four months. Functional evidence of recovery always precedes the roentgenological evidence.

The prognosis is not generally serious provided efforts are made to maintain the patient's alimentation by gavage or other means during the paralytic phase. Paralysis is merely one manifestation of the disease and is not necessarily

fatal of itself. Recovery depends upon the success of the treatment of the disease by antitoxin and other measures.

Paralysis Due to Botulism

The manifestations typically evolve in stages. There is usually a latent period lasting four or five days characterized only by certain prodromal digestive disturbances with nausea and vomiting of undigested food. It is typical of botulism that violent disturbances of the gastrointestinal tract do not usually occur. In this respect it differs from other forms of food poisoning.

The characteristic neuromuscular symptoms appear rather suddenly. At first there is dysphagia with a painful dryness of the throat, obstipation, and finally ocular paralysis affecting both the ciliary and the extrinsic muscles at once. The dryness of the throat is characteristic of botulism and is usually a premonitory sign of the esophageal paralysis in this disease. The dysphagia which is the result is usually described in the initial period of the infection. The mucosa of the throat and mouth is red, dry, and shiny. The secretions are profoundly altered. Difficulty with swallowing due in part to lack of secretions makes the ingestion of both liquids and solids laborious. But the element of paralysis is important as well. Paralysis of the soft palate is fairly frequent.

The ROENTGEN EXAMINATION usually demonstrates esophageal paralysis as a part of the inhibition of the motor function of the gastrointestinal tract. There is also evidence on fluoroscopy of paralysis of the constrictor muscles of the pharynx.

The dysphagia is sometimes so severe that swallowing is impossible and neither solids nor liquids will go down. The mechanism of this dysphagia has nothing in common with that of other conditions. It is the esophagus alone which is responsible for the aphagia. The barium mixture stops at the upper extremity of the organ and no peristaltic activity of any sort develops.

The origin of the paralysis of the esophagus in botulism is doubtless a central lesion limited to the cells which give rise to the motor fibers which supply the digestive tract. The lesion is confined to the nucleus.

Evidence that the toxin of botulism has a particular or selective action upon the cranial parasympathetic innervation is therefore the extreme dryness of the mouth and the esophageal dysphagia. The muscles of the pharynx, particularly the inferior constrictor, may be selectively involved as well.

TREATMENT Treatment is partly specific, partly symptomatic. Antibotulism serum or antitoxin is available. Pilocarpine may alleviate some of the dryness of the mouth. Administration of infusions of glucose solution with vitamin supplement is helpful as a temporary measure. When the aphagia is complete and unrelenting, feeding by gavage may become necessary.

CHAPTER 11

Diverticula

THE TERM *diverticulum* is applied to a circumscribed pouch or sac of variable dimensions developed at the expense of the wall of the esophagus and with a cavity which opens into the lumen. The condition was recognized many years ago by the older pathological anatomists particularly Ludlow (in 1767) by Cruveilhier, and by Zenker.

There are two types usually described according to the classification proposed by Zenker as (1) pulsion diverticula and (2) traction diverticula. This terminology is useful in explaining the difference between the types on an etiological basis, but it is not entirely satisfactory because it fails to take into account the basic anatomical and pathological differences between them.

A true diverticulum of the esophagus is actually a hernia consisting of a pouch of mucous membrane which protrudes through a weak spot in the esophageal musculature and, as it enlarges, gradually becomes elongated to the extent where it usually descends below the point of its emergence. It always occurs above a segment of the esophagus which has a sphincteric action. Thus the only two locations where a true diverticulum may be found in the esophagus are at the pharyngo-esophageal junction above the cricopharyngeus muscle and in the epiphrenic segment proximal to the muscle fibers which by reason of their peculiar intrinsic innervation previously described maintain the function of a sphincter at the lower end of the organ. A useful designation for diverticula of this type therefore is *juxtasphincteric diverticulum*.

The type which is usually called a traction diverticulum is in fact not a true diverticulum at all. These are localized distortions, angulations or funnel-shaped bulgings of the full thickness of the wall of the esophagus produced by the effect of an adherent inflammatory lesion in the periesophageal lymph nodes almost invariably at the level of the major bronchus or the bronchial carina. The usual etiology is tuberculous lymphadenitis. These pseudodiverticula occurring in the middle of the thoracic portion are therefore *nonsphincteric* in location and their mechanism of production is different.

Figure 200 shows the frequency of occurrence of the types and locations of diverticula of the esophagus in a personal experience of patients subjected to operation (R H S)

FREQUENCY OF OCCURRENCE OF THE TYPES OF
DIVERTICULA OF THE ESOPHAGUS

Juxtaspincteric (pulsion)	109
Pharyngo esophageal	94
Epiphrenic	15
Nonspincteric (traction)	9
Total	118

FIGURE 200 Table showing the relative frequency of occurrence of the types of diverticula

True Juxtaspincteric Diverticula

The mechanism which explains the development of a true diverticulum as recognized by Zenker consists of the effect of pressure upon an actual or potentially weak area in the wall of the esophagus, the pre existence of which, probably on a congenital basis, must be assumed. The weak point at the upper end is a triangular-shaped thin area in the midline of the posterior wall between the lower portions of the oblique fibers of the inferior constrictors of the pharynx just above the cricopharyngeus muscle. In the lower esophagus the potentially weakened area lies several inches above the cardia, just proximal to the sphincter-like segment and on the right lateral wall. The location of this defect on the right side is explained by the fact that the lower esophagus during embryological development becomes rotated partially in a counterclockwise direction as the stomach assumes its final adult position. The weak area, therefore, like that in the hypopharynx, was originally on the posterior wall.

Whether or not an unusual amount of intraluminal pressure is required to produce a diverticulum in a given patient is uncertain. It has been assumed that some disturbance in the synergic function of the muscles of propulsion above and the sphincteric segment below is a necessary prerequisite. Thus in the hypopharynx, if the cricopharyngeus muscle should fail to open or should open incompletely during the phase of contraction of the muscles of the pharynx, an abnormal degree of intraluminal pressure would be exerted against the potentially weak spot. This would tend to provoke a protrusion or herniation of the mucosa which, as it enlarges, would gradually produce a diverticulum.

It is of great clinical importance to take note of the fact that in a certain number of patients particularly elderly women, there is a coexisting upper esophageal web at the mouth of the esophagus. It is possible that the chronic dysphagia resulting from such a partial obstruction of the esophageal lumen may play, at least in part, a significant role in the development of the diverticulum in these patients.

The same mechanism can be envisaged to explain the development of the epiphrenic juxtaspincteric diverticula. A failure of coordination between the advancing peristaltic contraction of the esophagus above and the essential relaxation of the sphincter-like mechanism below would give rise to an increase in the intraluminal pressure at the level of the weak area as the force of the peristaltic wave is felt.

Although cases are on record of the occurrence of true pulsion diverticula in infancy and childhood, they are usually first recognized in adult life, principally in the later decades. This is doubtless because of the weakening of the tissues as a manifestation of senescence and the well known fact that incoordination of the muscles participating in the swallowing mechanism occurs more commonly in older people than in the young.

DIVERTICULA OF THE PHARYNGO ESOPHAGEAL SEGMENT

Of all true diverticula of the esophagus and pharynx, those occurring in the pharyngo-esophageal junction just above the cricopharyngeus muscle are the most common. The generally accepted frequency is 90 per cent. They are more common in men than in women (approximately two to one). The age of recognition of the condition varies from 35 to 80 or more years. The majority are first seen after the age of 50. Rarely they may be observed in children.

The pouch always emerges through the previously mentioned triangular-shaped weak area in the midline above the cricopharyngeus muscle (Figs. 201 and 202). As it enlarges, it descends in the prevertebral space between the pharynx, the esophagus, and the spine to reach sometimes as far as the superior mediastinum. As the sac grows larger, it usually tends to swing slightly to the left (Fig. 201). Rarely the point of emergence may appear to be on one side instead of in the midline in back, but this is usually caused by rotation as the sac enlarges. The pressure of a unilateral enlargement of the thyroid (goiter) is the most frequent cause of this variation.

The size of the sac varies, of course, depending upon its age and the amount

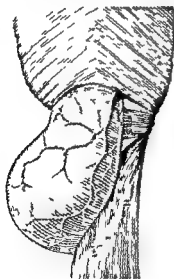


FIGURE 201

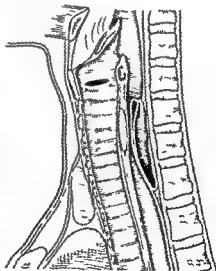


FIGURE 202

FIGURE 201 Drawing of a pharyngo-esophageal diverticulum emerging from the upper weak point above the cricopharyngeal sphincteric muscle.

FIGURE 202 Drawing of a sagittal section showing the characteristic location of a pharyngo-esophageal diverticulum behind the esophagus, its neck just proximal to the cricopharyngeus muscle. Note (1) the compression of the esophagus, (2) the almost direct continuity with the pharynx demonstrating the reason why such a diverticulum fills so readily.



1



2



3



4



5



6



7



8



9



10



11



12



13



14



15



16



17



18

Chen J. J. et al.

(See facing page for legend)

PLATE II

ESOPHAGOSCOPIC VIEWS OF PATHOLOGICAL CHANGES IN THE ESOPHAGUS MADE FROM LIFE (DISTAL ILLUMINATION)

(Photographic reproductions of oil paintings by Chevalier Jackson)

1 Orifice of an enormous diverticulum of the hypopharynx filled with barium. The patient aged 70 years was in a state of imminent asphyxia due to compression of the trachea. The dyspnea disappeared immediately after esophagoscopy aspiration of the barium.

2 The same patient after cleansing of the pouch. The orifice of the diverticulum is at the left. To the right in the upper quadrant of the field is the opening into the esophagus below the diverticulum. In between is the groove caused by the contraction of the cricopharyngeus muscle.

3 After one stage excision of the diverticulum in the lower part of the field is the esophagoscopy appearance of the closed-over orifice of the diverticulum.

4 Ulcerated gumma of the esophagus involving the thoracic portion in a man aged 32 years. Specific treatment cured the ulcer leaving a soft cicatrix with a tendency to produce stenosis. Dilatation was recommended.

5 Same case as 4 after a lapse of three years. The patient who was a sailor returned to the Clinic because when he ate quickly the food seemed to stop along the way before reaching the stomach. Esophagoscopy inspection showed a dilatation of the thoracic esophagus as shown here. At the left is the orifice of the stricture. At the right is a depression which was thought to be an incipient diverticulum. The whitish furrow between is a cicatricial band.

6 Same patient seen two years later with marked dysphagia for solids. The esophagoscope demonstrated a stricture (at the left) as well as a well developed diverticulum 4 cm. deep. Dilatation of the stricture through the esophagoscope ameliorated the swallowing difficulty but at the last examination the diverticulum had not changed.

7 Spindle cell epithelioma (Grade IV Broders) of the cervical esophagus in a man 66 years of age. The fungating growth lies beyond the cricopharyngeal fold.

8 Small diverticulum (about 3 cm. in depth) of the thoracic esophagus in a woman aged 55 years. The orifice of the diverticulum is at the right of the cicatricial band. A small whitish slightly raised scar is visible at the left. On the basis of the patient's history this is probably the result of an old peptic ulcer.

9 Spindle-cell epithelioma (carcinoma) of cauliflower type (Grade IV Broders) at the esophageal hiatus in a man aged 64 years with a complaint of dysphagia and obstruction at frequent intervals since the age of 50 years. There were according to the patient's history numerous previous exacerbations of peptic esophagitis but no proof of this had been obtained as the patient had never been subjected to esophagoscopy examination before his admission to the Clinic.

10 Normal esophageal hiatus as it appeared on esophagoscopy examination following the extraction of a foreign body (in this case a bone) lodged in the cervical esophagus in a man aged 25 years who had the habit of rapid eating.

11 Normal esophagus of the same patient one year later. The appearance is normal on inspection after the removal of a second foreign body. This time a mass of meat with perosteum and cartilage which had become lodged in the cervical esophagus while the patient was drunk.

12 Esophageal hiatus of the same patient as in 10 and 11 examined at the Clinic after 2½ years of military service with three months at the front. There had developed a typical esophagitis with achalasia but without appreciable dilatation.

13 Pedunculated proliferative carcinoma of the midesophagus in a man aged 31 years. The patient who had suffered dysphagia for two years attributed the sudden onset to the fact that he had swallowed a large piece of apple. Cyanosis of the mucosa and dilatation of the vessels result from imminent asphyxia due to obstruction of the trachea by neoplastic invasion. Biopsy showed an adenocarcinoma.

14 Ulcerated carcinoma of the lower third of the esophagus in a woman aged 49 years. Numerous grayish points are seen at the bottom of the ulcer. The partial obstruction of which the patient complained had begun one year before her admission to the Clinic. Histological study revealed a spindle-cell carcinoma (Grade III Broders).

15 Carcinoma of the cervical esophagus at the cricopharyngeus with a solid appearance and an indurated infiltrated base easily palpated by means of the esophagoscope. Biopsy showed a highly malignant atypical spindle-cell carcinoma (Grade IV Broders).

16 Undifferentiated infiltrating spindle-cell carcinoma with an indurated base palpable with the esophagoscope. Diagnosis confirmed by biopsy.

17 Ulcerated colloid carcinoma at the hiatus. Fungating projections and whitish points are visible in the depths of the ulcer. The hard infiltrated base could be felt easily with the esophagoscope.

18 Pedunculated fibrolipoma (benign) attached to the wall of the lower third of the esophagus. The base of the pedicle is exposed for esophagoscopy excision. The tumor 4 cm. in length was easily removed through the esophagoscope. No evidence of recurrence was seen on esophagoscopy examination four years later.

of its contents. When discovered early it may be merely a bulge. In patients who have had the condition for many years the sac may be enormous, holding as much as a normal stomach. The large sacs descend into the posterior mediastinum but have never been known to reach below the tracheal bifurcation. They are never adherent to the surrounding tissues unless there has been a complicating inflammation of the sac (diverticulitis) or of the esophagus with periesophageal phlegmonous mediastinitis.

The orifice of the sac varies from patient to patient as to its size and shape. As a general rule, up to the point where it reaches unusually large proportions, the size of the opening keeps pace with the increase in the size of the sac itself. A large diverticulum with a small opening is a rare occurrence. Often as the pouch becomes large, the diameter exceeds that of the esophagus, and as the diverticulum descends further and further, enlarging as it goes, the disparity in size between the mouth of the esophagus and that of the sac becomes very striking (Fig. 203, 3). With the smaller diverticula the opening is usually elliptical, lying behind and above the mouth of the esophagus with its long axis in the same direction as the pharynx. When the pouch becomes larger, however, the shape of the orifice changes to assume that of a transverse ellipse lying behind the mouth of the esophagus at the same level (Plate II). These observations are important when it comes to the surgical excision of a diverticulum in this location (see below). Occasionally the muscle layer of the pharynx forms a ring around the neck of the sac.

In rare instances the diverticulum may develop intramurally. The sac in such a case protrudes through the deeper muscle layer but, since the outer layer is firm and offers resistance, it dissects downwards between the layers. In this manner a sac of relatively small size may give rise to severe symptoms. The coincidence of unusually pronounced obstructive symptoms with a disproportionately small pouch, as disclosed at roentgen examination, should give rise to the suspicion that the diverticulum has developed intramurally. If this possibility is not known to the surgeon, he may fail to find it at the operation performed for its removal.

The diverticulum consists of a thin layer of mucous membrane covered by a filmy outer layer of connective tissue which is continuous with the same layer overlying the pharynx. Although the major portion of the pouch has no muscle covering, there is often a thinned out extension of the muscle fibers of the pharyngeal wall around its base, as though a portion of the muscle layer were pulled out upon it as the diverticulum enlarges. Running in the fascial layer which surrounds the mucosal herniation are a number of small arteries and veins which are continuous with those supplying the wall of the pharynx from which they emerge. These, of course, must be ligated and divided when the sac is removed surgically.

There are three principal stages in the development of a pharyngo-esophageal diverticulum. In *Stage 1* a small bulge comprises the beginning of the herniation but has no actual sac or pouch (Fig. 203, 1). This is analogous to the bubonocoele stage in the evolution of an inguinal hernia. In some persons the defect never enlarges beyond this incipient stage. In *Stage 2* the sac has actually emerged from the pharynx and, having extended downward, remains

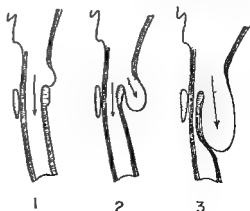
in position between the esophagus and the spine. In this stage the orifice of the sac has begun to tilt and change its shape as mentioned above (Fig. 203, 2). This is like the inguinal hernia which protrudes through the external ring but does not reach the scrotum. As Stage 3 is reached, the sac has become very large, extending into the mediastinum. Its orifice has completed its change of position to the horizontal and has reached its maximum dimensions. The esophagus meanwhile has become compressed by the pressure of the diverticulum which in this position fills first before any food can enter the normal passageway. This is analogous to the inguinal hernia which has reached the final stage of its enlargement with the sac full of omentum or intestines, filling the scrotum and pushing the testicle aside (Fig. 203, 3).

Clinical Characteristics

In the early phases of its development (Stage 1), there are no truly characteristic symptoms of a pharyngo-esophageal diverticulum. There may be, however, a period of many years' duration when the patient experiences vague discomforts which are actually premonitory manifestations, although the diagnosis is rarely suspected. Making allowances for the inevitable variations from one person to another, these may be: burning discomfort in the throat; pharyngeal tenesmus accompanied by choking spells; slight sticking pains; a feeling of tightness or constriction in the neck; dry cough; and intermittent dysphagia attended by occasional aspiration of material into the air passages. Sometimes there is exaggerated salivation or a malodorous breath. The diagnosis of dry pharyngitis or of pharyngeal paresthesia is sometimes made before the existence of the diverticulum is suspected.

It is only after many years have elapsed that the pouch reaches a size large enough to cause the characteristic symptoms which begin with Stage 2. This coincides with the time when the diverticulum begins to hang down and thus to accumulate food, liquid, and saliva as a part of what the patient has normally swallowed. Now the patient begins to experience a sensation of stoppage during deglutition of either liquids or solids. This is felt in the neck or upper chest. It is caused sometimes by the arrest of food in the pharynx at the opening of the pouch. More often it means that the diverticulum, when it becomes distended with material swallowed, produces an actual obstruction by pressure

FIGURE 203 Diagram demonstrating the three stages of the development of a pharyngo-esophageal diverticulum. Stage 1 Beginning herniation; no actual sac. Stage 2 Well established hernia with protruding pouch; the orifice of which has begun to turn horizontally; slight pressure on the esophagus. Stage 3 Large pouch extending downward; its orifice almost horizontal and much larger than the mouth of the esophagus; marked compression of the esophagus below.



against the esophagus. In the third stage of the disorder this aspect assumes large proportions, and on reaching this stage many patients who have previously neglected the condition become malnourished or even cachectic because of continuous, almost total obstruction.

The majority of patients learn how to evacuate the pouch by washing it out with mouthfuls of water, by pressing on the side of the neck, by bending the head from side to side, or by stooping over. Some acquire the habit of eating while lying down, others even ruminate. With a few patients the pouch fills so slowly that it may not cause enough pressure upon the adjacent esophagus to require evacuation of its contents more than once a day or once every two or three days. When this is the case, it is characteristic for the patient to bring up remnants of food eaten a long time previously. The material in this instance may be partially decomposed and malodorous.

Another symptom which is often annoying and embarrassing for the patient is the occurrence of gurgling noises heard during deglutition, particularly of liquids, because of the admixture of air. In fact, with many patients this is so characteristic that requesting a patient suspected of having a diverticulum of this region to swallow a glassful of water in one's presence is sufficient to clinch the diagnosis without other evidence. Sometimes after the patient has swallowed some water, if he is instructed to hold his breath with the glottis closed and shake his head, a slopping noise can be heard in the pouch. Another diagnostic maneuver which sometimes helps is to have the patient fill the sac with air by making several gulping motions, whereupon pressure with the fingers over the sac will cause the air to burst forth into the pharynx with a characteristic noise.

Röntgen Examination

On giving the patient a swallow of liquid barium, the diverticulum appears as a pouch suspended from the esophageal wall by a pedicle (Fig. 204). The pouch fills with the first swallow and the barium collects in the lower portion where it produces a horizontal fluid level often surmounted by residual fluid contents or air. The lower portion of the diverticulum has rounded contours which may press the esophagus forward (Fig. 205).



FIGURE 204. Serial roentgen films of a patient with a pharyngo-esophageal diverticulum made during deglutition. 1 First swallow of barium fills the diverticulum but very little enters the esophagus. 2 Second swallow with the diverticulum already full, the barium passes down the esophagus. 3 4 Barium remaining in the diverticulum after the esophagus has emptied itself.

FIGURE 205 : Film showing a pharyngo esophageal diverticulum filled with barium and producing marked compression and deviation of the adjacent portion of the esophagus. Note deviation of the diverticulum to the left (Stage 3)



Because of the absence of a muscular layer which might expel it the barium tends to remain indefinitely in the interior of the pouch unless the patient can evacuate it by some technique of his own. Sometimes because of muscle incoordination of the pharynx and larynx during deglutition a portion of the barium is aspirated into the trachea giving the impression of a fistula (Fig. 196)

Esophagoscopy

In general it is unwise to esophagoscope patients known to have a pharyngo-esophageal diverticulum unless a foreign body must be extracted. The examination is dangerous because the walls of the sac are so thin. The end of the instrument usually tends to enter the orifice of the pouch as though it were a continuation of the pharynx. There is often a fold of mucosa pale in color located at the orifice. The walls of the sac are usually lined with debris. The mucous membrane is likely to be hyperemic with erosions and superficial ulcerations. Sometimes the vestiges of a former inflammation can be recognized in the form of cicatricial bands (Plate II 1 2 and 3)

After the sac has been explored the instrument should be withdrawn part way and a search made anteriorly for the cleft which corresponds to the mouth of the esophagus. This part of the examination is always difficult and very delicate because of the invariable presence of spasm.

Sometimes the reverse situation prevails. The orifice of the diverticulum is narrow, looking like a fissure or somewhat like the anal orifice. The tube slides by it without stopping and if the intention is to explore the pouch a considerable search must be made in order to find it. Sometimes it is better to use the Jackson esophageal speculum than the usual full length instrument.

Differential Diagnosis

The clinical triad of regurgitation of food eaten days before, the continual presence of an abundance of frothy mucus in the pharynx, and the occurrence of gurgling noises in the throat during deglutition is so characteristic that there is hardly any chance of failing to make the diagnosis of a pharyngo esophageal diverticulum on the part of one who is well informed. This syndrome should at least cause enough suspicion to prompt the performance of a roentgen exam



FIGURE 206 Esophagogram showing in *A* a shadow above a stricture which might be interpreted as due to a diverticulum in *B* a true lateral view the shadow is seen to be due merely to a dilatation of the esophagus above the stricture

ination for confirmation. The presence of frothy saliva in the pyriform sinuses alone, however, is not diagnostic because of the frequency with which this sign is observed in other abnormalities, particularly neuromuscular disturbances with functional dysphagia.

A word of caution should be mentioned regarding the interpretation of roentgen films in certain instances. Unless a lateral film is available, it is relatively easy to mistake the dilated cervical esophagus proximal to an inflammatory stricture or even a scirrhus form of carcinoma in the lower cervical segment for a pharyngo esophageal diverticulum. In the lateral view the true diagnosis becomes clear (Fig. 206).

Complications

The small pouches rarely cause any trouble except for the unusual occurrence of diverticulitis due to infection secondary to erosion by an impacted foreign body such as a piece of bone or a fragment of lobster claw. The large

sacs, when they remain filled with food which the patient is unable to evacuate may cause serious difficulties. The emaciation resulting from the inability of the patient to swallow, due to the effects of distortion of the upper end of the esophagus and the compression by the diverticulum below, has been mentioned above. Other effects from an overdistended large intramediastinal diverticulum are dyspnea due to tracheal compression, obstruction of the venous return from the head and neck by pressure on the innominate veins and vena cava, and the toxicity and local reaction due to infection arising from the decomposition of diverticular contents. Recurring bronchitis, aspiration bronchopneumonia, atelectasis, and lung abscess are not infrequent complications. A lung abscess due to aspiration of partially decomposed food is putrid, with a fetid odor and full of anaerobic organisms. The most frequent location is in the lower lobe of the right lung. The malnutrition conjoined with the effects of toxic absorption may be responsible for polyarthritis and facial brachial, or thoracic neuralgia. As a result of esophagospasm, pseudo-angina pectoris may be experienced because of reflex irritation of the vagus nerves.

A relatively unusual complication is the development of a carcinoma in the pouch, or more usually at its orifice. Opinions differ as to the frequency of this association. A frequently quoted figure is 10 per cent of the cases, but this is probably much too high except in neglected cases of long duration.

One of the most serious complications of large, long neglected diverticula is infection and necrosis of the wall of the pouch, which may result in perforation and the spread of sepsis throughout the neck and mediastinum. The usual site of perforation is in the bottom of the sac where the decomposing contents stagnate. This occurrence is occasionally the cause of death in emaciated poorly nourished patients whose resistance is low because of starvation.

Even in patients with smaller diverticula a form of diverticulitis may develop and lead to peridiverticular fibrosis and adhesive fixation which increase the technical difficulty of their removal.

Prognosis

In the average instance, diverticula of the pharyngo-esophageal junction are important only because they are a nuisance and often a source of embarrassment to the patient. In the large, neglected diverticula continuously full of septic material, however, the prognosis is always grave.

Treatment

NONSURGICAL. Although there is no medicinal treatment for this condition it is possible to ameliorate the symptoms, in the case of the smaller diverticula at least by attention to diet and habits of eating, sometimes supplemented by efforts to dilate the mouth of the esophagus. So far as diet is concerned the food should be soft including thick soups, cooked cereals, and purees with generous amounts of butter. Oils and fats pass easily. Fruits containing seeds, nuts, salads, spices, and wine and other alcohol containing liquids must be forbidden. Each patient, however, must learn by experience how to regulate his diet to the best advantage.

The method of eating is also important. Mastication must be slow and

prolonged to favor adequate admixture with saliva. The meal should be unhurried. It is wise for the patient to lie down after meals. Postcibal evacuation of the pouch should be practiced if a method agreeable to the patient can be found. The physician can sometimes make helpful suggestions in this respect. Sometimes the patient can learn to wash out the pouch with repeated mouthfuls of water which he then regurgitates.

If the case is not of too long standing and the diverticulum not too large, the difficulties may be ameliorated and the pouch may even shrink in size as a result of systematic dilatation of the cricopharyngeus muscle. This may be done under direct vision through the esophagoscope or by the multiple bougie technique previously described (Chapter 5). All such manipulations demand diligence and skill on the part of the operator, and should never be attempted by the inexperienced. Dilatation of the mouth of the esophagus is most often applicable to the treatment of patients whose advanced age or poor general condition are such that surgical excision is contraindicated.

SURGICAL TREATMENT Whenever possible, surgical excision (*one-stage diverticulectomy*) is the ideal treatment. The indications for operation depend upon the case, but by the time the patient begins to experience definite symptoms he is usually anxious to be relieved of the difficulty. Even if he is not, excision should be recommended because of the well known tendency of these diverticula to grow larger progressively as the patient advances in age. When the pouch is large, the indications for removal are obvious if not imperative.

Preoperative preparation should include whatever measures may be necessary to improve the patient's state of health to the optimum point. If the patient's teeth are in poor condition, they should receive proper attention before proceeding.

If possible, the sac should be emptied, washed out, and then kept empty of solid material by prescribing a liquid diet during the forty-eight hours preceding the operation. During this period, also, the patient should be requested to gargle and wash out the pouch with a solution of streptomycin (0.25 gm. per 100 cc.) every four hours during the daytime.

General anesthesia administered through an intratracheal tube is usually employed. The type of anesthesia and the skill with which it is administered are of great importance in the avoidance of postoperative complications, especially a disruption of the sutured pharynx from retching or vomiting. A satisfactory technique is the use of intravenous Pentothal for induction of the anesthesia, with maintenance of analgesia by means of inhalation of nitrous oxide admixed with oxygen and injections of curare derivatives or succinylcholine in small doses to produce what relaxation may be required.

If there is any doubt about the wisdom of using general anesthesia, however, the operation can be performed easily with local and regional infiltration of procaine hydrochloride solution (1 per cent).

Technique Although a curved transverse incision similar to that used for a thyroidectomy may be employed, the most convenient approach is through the left side of the neck. If the surgeon happens to be left handed, however, he will find that the placing of sutures in the hypopharynx can be more easily accomplished through the right side. Because the pouch arises from the midline

even though it may deviate to one side or the other, usually to the left, the choice of side depends only upon the convenience of the operator

The incision is made along the anterior margin of the left or right sternocleidomastoid muscle from a point just above the inner end of the clavicle to a point about 2 or 3 cm below the angle of the jaw (Fig 207) As the platysma muscle is divided, a sensory nerve which is a branch of the cervical plexus is encountered crossing the upper portion of the incision This should be avoided if possible because if it is severed, anesthesia of the triangle of skin between the upper third of the incision and the anterior half of the lower border of the mandible will result

The dissection is deepened through the fascial plane in front of the muscle and carried down in front of the carotid sheath behind the left lobe of the thyroid into the prevertebral fascial space At this point it is frequently necessary to ligate and divide the superior thyroid artery and vein as they cross the operative field In some instances it may be necessary also to divide the omohyoid muscle to improve the exposure in the lower end of the incision (Fig 208) The small artery and vein and nerve which supply this muscle are always divided Division of the loop of the ansa hypoglossi nerve is a common occurrence No functional disturbance results from this By blunt dissection with the scissors the prevertebral fascial space is opened widely both up and down and sideways, thereby exposing the posterior surface of the pharynx and esophagus to view The diverticulum can then be recognized easily and unless surrounded by adhesions from previous diverticulitis or unsuccessful attempts to remove it

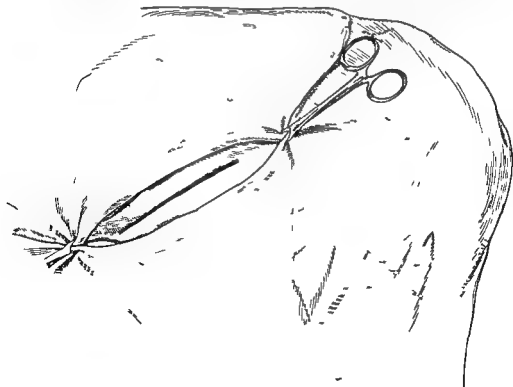


FIGURE 207 · Incision for the removal of a pharyngo-esophageal diverticulum

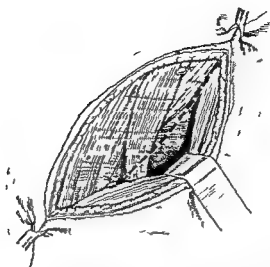


FIGURE 208 Deepened incision showing muscle layers. The broken line indicates the direction and extent of the dissection for the exposure of the esophagus. Note: The omohyoid muscle which crosses the field must be either retracted or divided.

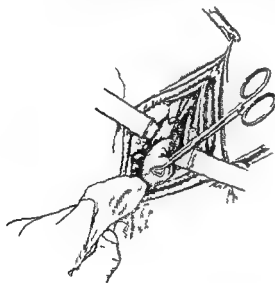


FIGURE 209 Freeing of the diverticulum by blunt dissection.

the pouch can be drawn forth from its bed with a grasping forceps (Collin type) (Fig. 209).

At this point in the procedure the anesthetist must be prepared to empty the pharynx by aspiration because, if the sac is full its contents will be expelled. It is for this reason also that a cuffed intratracheal tube must be used for anesthesia in order to prevent the diverticular contents from passing down the trachea into the lungs. If the operation is done with local anesthesia however, the integrity of the patient's reflexes insure him against this danger.

In certain instances the access to the diverticulum may be obstructed by a goiter involving the left lobe of the thyroid, but this obstacle is readily overcome by excising the tumor before attempting to free the pouch.

As the diverticulum is drawn up through the incision a layer of connective tissue between it and the esophagus is divided. In preparation for its removal

the diverticulum must be freed close to its base by circumcising the outer fascial layer down to but not yet through the mucosa. In doing this a thin layer of muscle fibers spreading out onto the base of the sac may be observed, as mentioned above. In these extramucosal musculofascial tissues, several small branches of the pharyngeal arteries and veins must be severed as they branch out upon the diverticulum forming its blood supply. In order to make a good closure after the excision of the sac, it is necessary to preserve an adequate amount of muscle tissue along the edges of the defect. To accomplish this the circumferential incision through the outer layers must not be made too close to the base of the sac. This can be done by releasing the pull upon the sac from time to time as the dissection is in progress (Fig. 210).

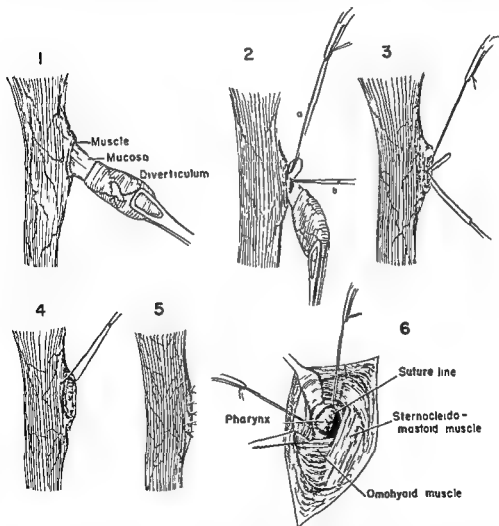


FIGURE 210. Steps in the actual removal of pharyngo-esophageal diverticulum. 1. Outer layer has been circumcised exposing the mucosal layer of the pouch. 2. Start of removal showing the initial incision in the mucosa with one suture tied (a) and ready to be cut as the second suture is tied (b). 3. Diverticulum removed, last few mucosal sutures being inserted. 4. Start of the closure of the muscle layer. 5. Closure completed. 6. Relations of the structures in the field of operation: site of removal of the diverticulum at the bottom. Note: The suture line will rotate into the midline posterior position as soon as the traction sutures are released.

Once the mucosa has been exposed around the entire circumference, the removal of the diverticulum is begun. This is usually best accomplished in the long axis of the pharynx. There are instances, however, in which the line of excision and closure can be made with less possibility of constricting the lumen or distorting the wall of the pharynx by proceeding in the direction of the circumference. A small opening is made at the end where the removal is to begin and a suture of 5-0 silk is inserted in such a manner as to leave the knot on the inside after it is tied (Fig. 210, 2). This is held by an assistant as the second suture is placed. As the strands of the second are pulled up for tying, those of the first which have already been tied are cut. The incision through the mucosa is then carried a little further on each side of the sac and the next suture is inserted. This process is continued until the sac has been removed (Fig. 210, 3). It is important from time to time during the process to release the traction on the sac to make certain that some of the pharyngeal mucosa is not being excised along with the sac. This would of course lead to contraction of the lumen after the defect is closed.

Another important precaution should be mentioned. Because a mucosal web at the mouth of the esophagus is a fairly frequent occurrence (4 per cent of patients operated upon), especially in elderly women, an instrument or sometimes the surgeon's finger should be passed into the esophagus through the opening at the base of the diverticulum as its removal is being carried out. If a web is encountered, it should be divulsed or if necessary actually excised surgically. In order to excise it, it may be necessary to enlarge the opening by a downwards extension through the posterior wall of the upper esophagus. The web is then trimmed away with the scissors, and the mucosal edges where it was attached are approximated with sutures of fine silk. The incision of the esophagus is closed in two layers with the same material. The defect resulting from the removal of the diverticulum is then closed in the usual manner.

After the pouch has been removed and the last mucosal suture tied and cut, the muscle edges are approximated with sutures of the same material, and finally an outer layer of mattress sutures in the fascial covering of the pharynx is used to invert the closure slightly and provide further reinforcement (Fig. 210, 4, 5).

The closure of the muscle layer is usually performed in the long axis of the pharynx, but if the mucosa has been excised and closed circumferentially and in some instances even when the mucosal suture line lies in the lengthwise direction, it is best to close the muscle layer circumferentially. The same technique is used in either event.

The cervical incision is closed with a layer of interrupted 4-0 silk in the platysma muscle and another in the skin.

A drain should be inserted only in the rare instance when there is oozing which might lead to the accumulation of blood and serum. This is usually in patients who have had diverticular infection and inflammatory fixation.

Aftercare. Every effort should be made to avoid retching and vomiting. A proper choice of anesthesia and anesthetist is the best insurance against this occurrence. Adequate fluid intake is maintained at first and supplemented later, after oral administration is begun, by intravenous infusions of glucose or saline.

solution to which vitamins C and B complex may be added in patients whose nutritional condition is poor. After twenty-four hours, oral intake is started with 30 cc of water per hour taken in sips. The amount of liquid taken may be increased gradually from day to day, with daily changes in the character to include nourishing fluids. By the fourth or fifth day the intravenous infusions may be omitted. The patient should be cautioned to swallow slowly and in small mouthfuls. Soft solid food is begun cautiously by the fifth or sixth day and a regulation soft diet is reached by the time the patient is ready for discharge two or three days later.

Antibiotic medication, usually with penicillin and streptomycin, is continued for five days or until it is certain that no infection has occurred. The patient is allowed out of bed on the first day after operation and is usually ready for discharge from the hospital about one week later.

Complications. The mortality is nil and complications are few. Fistula formation is a rare occurrence. It results from retching or vomiting. Spontaneous closure is the rule. Temporary interference with deglutition may develop from edema and swelling at the site of closure of the defect resulting from the removal of the sac, especially if too much mucosa has been excised. As the surgeon's experience with the operation increases, however, this difficulty can usually be avoided.

An occasional patient may develop hoarseness as a result of paresis of the left vocal cord. This does not mean that the left recurrent laryngeal nerve has been injured, since it is almost never encountered in the performance of the operation. The cause is usually the pressure of a retractor against the main trunk of the left (or right) vagus nerve as the carotid sheath is held aside. This can be avoided if the surgeon is aware of the possibility. The tabulation (Fig. 211) illustrates the actual occurrence of complications after surgical excision in a series of seventy-five patients.

OUTMODED PROCEDURES. Because they are sometimes still mentioned as proper procedures in the treatment of a diverticulum of the pharyngo-esophageal junction, certain operations which should have been supplanted by one-stage diverticulectomy may be mentioned.

The first of these is *inversion of the diverticulum* so that it hangs within the lumen instead of exteriorly. This harks back to the days when surgeons were afraid to open the pharynx and when attempts to remove the pouch by the crude methods then in use—often ligation at the base and inversion of the stump—were

POSTOPERATIVE COMPLICATIONS
(75 Patients)

Fistula (spontaneous closure in 3 months)	1
Postoperative edema	2
Hoarseness due to	
Laryngitis (from intratracheal tube)	2
Vocal cord paresis (temporary)	3
Right	1
Left	2
Urinary retention	3
Cholecystitis	1
Deaths	0

FIGURE 211 Postoperative complications in a series of 75 patients operated upon for the removal of a pharyngo-esophageal diverticulum.

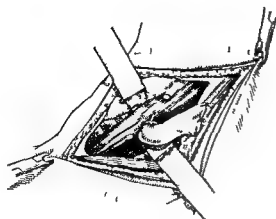


FIGURE 212 Diverticulopexy Drawing showing the apex of the diverticulum sutured to the sternocleidomastoid muscle in the upper angle of the incision

followed usually by disastrous failure. The operation has nothing to recommend it in the present day.

The next is *diverticulopexy*, where the sac is freed and sutured to the upper portion of the incision, which is then closed over it (Fig. 212). This procedure often eliminates the patient's symptoms. It has a limited place in the treatment of poor risk patients whose condition is so precarious that they might not survive the removal of the pouch. It can be performed easily with local anesthesia.

The third obsolete procedure is *two-stage diverticulectomy*. This was devised by C. Mayo in 1912 in an effort to improve the results obtained in that day with the gross techniques then in use. Like all two-stage operations, however, it represented a phase in the development of a procedure the ultimate form of which is the one stage technique already described. It has the obvious disadvantages of prolongation of stay in the hospital, increased discomfort for the patient, and a higher incidence of complications such as stricture formation and fistulization. It might still be employed in a poor risk patient.

DIVERTICULUM OF THE LOWER ESOPHAGEAL SEGMENT (EPIPHRENIC)

Diverticula occurring in the lower esophageal segment are not common (see page 248). As with the juxtacardiac diverticula, they are encountered usually in persons of middle age or beyond. They tend to be somewhat more common in men than in women. They may be small but when first discovered they tend to be larger than those in the upper region, often reaching a diameter of 9 to 10 cm. or more. They may develop at any level in the lower third of the esophagus, but the most frequent location is within the lower 10 cm. They usually emerge from the right side of the esophagus. In shape they are globular or formed like a bottle, a pear, or even like a mushroom. The orifice is either round or oval in shape. When ovoid the long axis of the opening is always the same as that of the esophagus (Fig. 213).

As with pharyngo-esophageal diverticula, those which develop in this region consist of a mucosal pouch which emerges through a place of separation in the muscular layer. They are covered by a layer of fascial connective tissue in which run several small arteries and veins which are branches of the esoph

FIGURE 213 Drawing of an epiphrenic diverticulum of the lower thoracic esophagus



ageal vessels. They lie free in the mediastinum without adherence to any adjacent organs or structures.

As mentioned before, the epiphrenic diverticula are justaxphincteric in that they emerge above the level of a portion of the esophageal musculature that has a sphincter-like function. They probably develop because of disturbances in the coordination or synergy between the mouth of the esophagus and this specialized musculature of the lower esophageal segment. In most instances there is doubtless a congenital weakness of the wall of the esophagus at the point where they develop, without which they would not occur.

Clinical Characteristics

At first the symptoms are vague. There may be a certain amount of dysphagia consisting of transitory sensations of food stopping or being held up temporarily in its passage into the stomach. The patient localizes this sensation in the lower part of the chest. As time passes, the patient begins to feel the symptoms of lower esophagospasm with a heavy or full feeling beneath the sternum or xiphoid process, or pain referred to the shoulders and neck along the pathways of the vagus nerves. These symptoms are usually felt only after eating. Aerophagia, nausea, bouts of coughing, or even simulated asthma, the result of reflexes mediated through the pulmonary plexus, may be observed.

As the diverticulum grows larger, usually over the course of many years, the patient begins to experience regurgitation of previously ingested food and liquids. Sometimes the material is mixed with blood, suggesting the presence of an ulcer. Usually the regurgitation occurs spontaneously, often on changing position as while turning over in bed. It may occur during sleep. Often the

patient finds that he has to avoid lying on one or the other side to prevent this occurrence

Finally the symptoms grow severe. The diverticulum when filled begins to cause obstruction of the esophagus from pressure or angulation. The patient may experience a choking sensation, with vertigo and faintness, relieved only by vomiting. In some instances he discovers means of avoiding overfilling of the pouch or of emptying it when full. These may be the ingestion of a glassful of hot water, movements of the chest, or pressure upon the abdomen. Such maneuvers are often described in intricate detail by those afflicted with the difficulty, just as in the case of patients with mega-esophagus.

The characteristics of the contents of the pouch vary depending upon the kind of food taken and the length of time it has been stagnant in the diverticulum. The odor is often bad and the breath of the patient is always unpleasant. Some times there are large amounts of mucus because of excessive secretory activity of the mucous glands within the sac.

Esophagitis may develop and the resulting secondary esophagospasm adds to the severity of the symptoms. Fever may arise if the element of infection is pronounced.

In addition to the purely esophageal manifestations, the patient may experience other disturbances of the gastrointestinal tract such as anorexia and attacks of diarrhea. Progressive emaciation may ensue, partly as a result of these and partly because of the obstructive dysphagia.

Furthermore, as a result of the disturbance produced by the continuously filled pouch, the vagus and sympathetic systems are affected and the patient may experience tachycardia, headache, hot flushes, vertigo, and weakness.

Roentgen Examination

The diagnosis of epiphrenic diverticulum is easily made by roentgen examination. When the diverticulum is small, however, it may produce so slight a shadow of so variable a shape that, without evidence of air and fluid level, it may pass unnoticed. At this stage the pouch may not fill at all in the erect posture. In fact, it may be seen only in the dorsal or ventral recumbent positions or in the Trendelenburg position. On the other hand, if a thick paste is given the material may stick always at one place, which should give rise to the suspicion that a diverticulum may be there. This should prompt the repetition of the examination.

When the diverticulum is of moderate or large size it shows up clearly as an obvious pouch attached to the wall of the esophagus. There is often a fluid level of barium in the bottom surmounted by air above it. The orifice can usually be recognized at the upper limit of the fluid level. If the mouth of the sac is unusually large and the sac dependent, no air may be seen due to the fact that the pouch fills completely with the opaque material (Fig. 214).

Esophagoscopy

Esophagoscopy is not necessary to make a diagnosis. It may be employed, however, to evaluate the condition of the esophagus itself in cases complicated by esophagitis or carcinoma. The examination is rather dangerous. In fact, if

the operator is not informed by previous inspection of the roentgen films of the probable location of the pouch, the end of the instrument may slip through the orifice without his becoming aware of the fact. A perforation can then be avoided only by delicate handling of the instrument which permits him to discern the resistance offered by the thin bottom wall of the sac. With a large pouch, the orifice also is usually large. The sac is often full of alimentary debris, but after aspiration the mucosa which is uncovered appears essentially normal. When esophagitis is present, the mucosa is hyperemic and edematous. With a diverticulum of long duration, shallow ulcerations or islands of scar tissue may be seen.

When the orifice is small, its discovery is sometimes difficult. The tube, as it smooths out the folds of the mucosa, discloses a slit or a circular opening which when pressed upon permits the escape of the remnants of food. The image seen through the tube is often reminiscent of the tracheal carina or the partition separating the two nasal fossae (Plate II, 8).

Differential Diagnosis

Without the benefit of roentgen examination it is impossible to establish more than a suspicion of the true diagnosis, although on the basis of the symptoms there can never be any doubt that the esophagus is the source of the



FIGURE 214 A B Roentgen films of an epiphrenic diverticulum above a hiatus hernia

patient's difficulty. The roentgen appearance is so characteristic that there is little likelihood of confusion, but brachyoesophagus and large hiatus hernias must be excluded. A hiatus hernia and an epiphrenic diverticulum may occur together (Fig. 214).

Complications

These have almost all been mentioned already. They include obstruction of the esophagus from pressure by a large distended pouch, and bronchopneumonia, pulmonary atelectasis, or abscess of the lung from aspiration of regurgitated contents. Local complications include infection of the wall of the diverticulum, sometimes followed by peridiverticulitis and mediastinitis, ulceration, and occasionally perforation of an area of necrosis or an ulcer in the diverticular wall. This almost invariably leads to the development of an abscess in the adjacent area of the mediastinum which, when drained to the exterior by mediastinotomy, eventuates in the formation of an esophagocutaneous fistula. Carcinoma has rarely been reported in an epiphrenic diverticulum.

Prognosis

The smaller epiphrenic diverticula are little more than a source of annoyance to the patient, but as such, because of the unrelenting nature of the symptoms, their removal often becomes advisable. The large ones, however, present an entirely different prospect and if not removed may lead to serious consequences from malnutrition, hemorrhage, perforation, and sepsis.

Treatment

There is no satisfactory medicinal treatment. Careful eating habits, the use of a soft solid and liquid diet, and sometimes the exhibition of antispasmodics such as tincture of belladonna may have a limited palliative value in the patients who have a relatively small pouch. When the pouch is large and in all patients whose symptoms warrant it, surgical excision should be advised.

SURGICAL EXCISION. Preoperative Care. The usual preparation of any patient about to undergo a thoracic operation is employed. If the hemoglobin level is low, one or more transfusions of blood are administered as required. A solution of streptomycin in water is given orally every four hours during the two days preceding the operation. If the patient is dehydrated and malnourished because of obstruction, the appropriate solutions with vitamin supplement (B complex and C) should be administered until the optimum point of treatment has been reached.

Before the patient goes to the operating room a Levin tube is inserted into the stomach in order to avoid overdistention and subsequent vomiting. It is a common occurrence, however, when the diverticulum is exposed at operation to find the end of the tube coiled up within it. The tube must then be withdrawn part way and redirected into the proper channel by the surgeon, whereupon it can be pushed into the stomach by the anesthetist.

General anesthesia administered through an intratracheal tube must of course be employed. Pentothal intravenously, nitrous oxide, and muscle relaxing agents is the combination preferred.

Technique The approach may be through either the right or left pleural cavity, depending upon the circumstances. If, as is usually the case, there is no likelihood that an esophagectomy will be necessary, the right side is somewhat better than the left because of the fact that in the majority of instances the pouch bulges to that side. If a resection is to be carried out because of a cicatricial stenosis or the presence of a carcinoma or if a coexisting hiatus hernia is to be repaired, the left side should be chosen. In the latter case it must be admitted a repair can actually be carried out through the right side but it is much more difficult to repair a hernia from the right, especially if it is a large one. As a matter of fact, so far as the diverticulum itself is concerned, the choice of side is relatively unimportant because of the ease with which the esophagus in its lower portion can be rotated during the procedure (Fig. 215).

A standard thoracotomy incision is made through the eighth intercostal space (Fig. 215). The mediastinal pleura overlying the lower esophagus is incised and the diverticulum is exposed to view. It is grasped with a Collin forceps and freed of its loose areolar tissue attachments. The neck of the sac is then circumcised and the diverticulum is removed using exactly the same technique as that employed for the extirpation of a diverticulum of the pharyngo-esophageal junction as already described (Figs. 216 and 217).

The mediastinal pleural edges are approximated loosely and a Foley catheter is inserted through a lower intercostal space to be put on closed suction drainage when the patient reaches his bed. The wound is closed in the customary manner with pericostal or percostal sutures of chromicized catgut and interrupted sutures of silk in the remaining layers.

In patients who have a diverticulum complicated by the presence of cicatricial stenosis from esophagitis, by an esophageal ulcer or by a carcinoma, or by a periesophageal abscess and fistula tract due to a perforation, a resection must be performed and followed in most instances by an esophagogastric anastomosis. This eventuality must be anticipated and a left sided approach employed. It should be pointed out in passing that the level of transection of the esophagus must in every case be made through normal esophagus well above the pathologic tissues. The anastomosis when completed should lie as far as possible away from the region where an abscess was.

Aftercare The usual measures employed after any thoracotomy are followed: frequent turning from back to operated side, early rising, usually on the second day, removal of the thoracic drainage catheter after forty-eight hours, etc. The Levin tube is left in place until the possibility of gastric distention is over, usually on the third or fourth day. This safeguards the patient against the danger of disruption of the esophagus at the closure site as a result of vomiting. Small amounts of fluid beginning with water (30 cc. per hour) are started on the day after operation, with a more rapid increase in amount and kind from day to day than in patients who have had an esophagectomy. Transfusions of blood and intravenous infusions of glucose solution supplemented with vitamin B complex and vitamin C are given in diminishing amounts until all alimentation is by the oral route (fifth to sixth day). The patient is discharged from the hospital on a soft solid diet which can be supplanted, within two weeks from the time of his discharge, by a normal food intake.

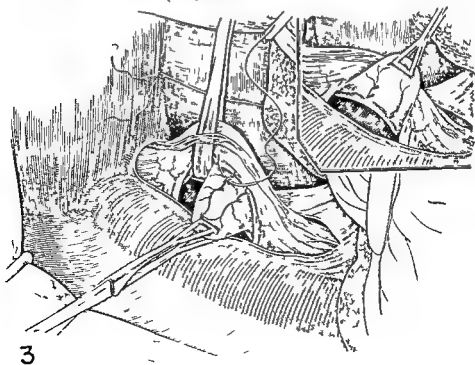
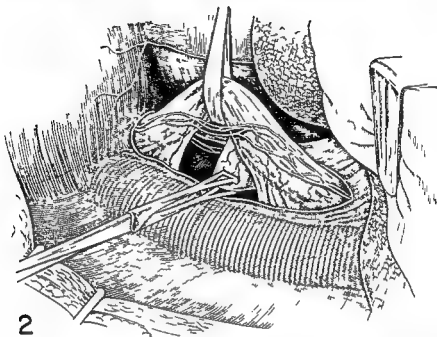


FIGURE 215 (See facing page for legend)

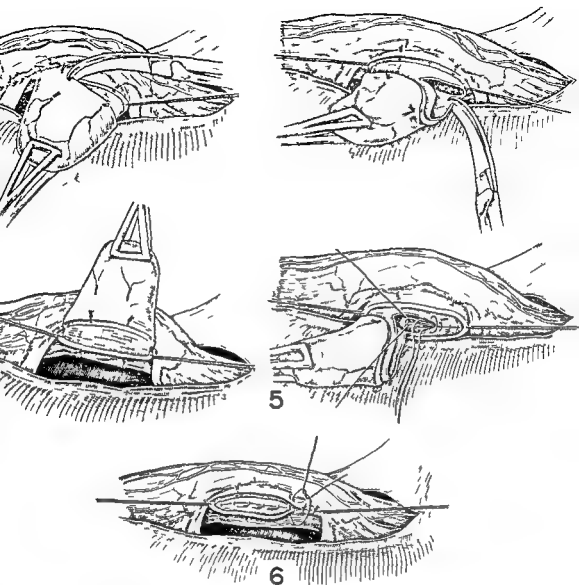


FIGURE 216

FIGURE 216 Further steps in the removal of an epiphrenic diverticulum 4 Circumcision of the outer layer exposing the mucosal lining 5 actual excision showing mucosa closure technique 6 mucosa closed muscular layer closure started (After J Perrotin)

Complications With the exception of the cases complicated by abscess formation in which trouble from infection may arise the recovery is usually uneventful. If the excision of the pouch and the closure of the defect have been performed correctly, leakage cannot occur. Pulmonary atelectasis or pneumonitis and other occasional complications are no more likely to develop than with any thoracic operation.

FIGURE 215 Excision of an epiphrenic diverticulum of the esophagus 1 Position of the patient and the line of incision (left thoracotomy approach) 2 exposure of the diverticulum in the lower mediastinum (Note: By rotating the esophagus the diverticulum which is attached on the right is drawn readily into the left chest for excision) 3 full exposure after rotation of the esophagus is completed (After J Perrotin)

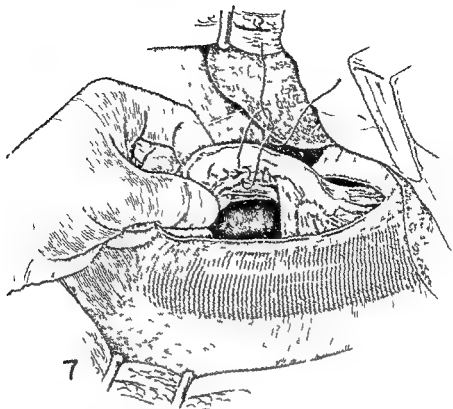


FIGURE 217 Completion of closure with an outer layer of Lembert sutures (After J. Pirogoff)

Nonsphincteric Pseudodiverticula

As explained above, these are not true diverticula either by the mechanism of their formation or by their anatomical characteristics. As will be appreciated from Figure 218, they are funnel shaped abnormalities or angular distortions of the full thickness of the wall of the esophagus produced at the site of adherent inflammatory tissues, usually chronic tuberculous lymphadenitis. The usual location is next to the subcarinal area or behind the bronchi where a large group of lymph nodes lies in direct contact with the esophageal wall. Because of bouts of infection in these nodes a considerable degree of periadenitis with marked inflammation of the surrounding areolar tissues occurs. This leads to periesophagitis and fixation of the esophagus to the mass of nodes by cicatricial adhesions. The esophagus loses its mobility and, little by little, as a result of the movement produced by deglutition, by respiration, or by bouts of coughing, its wall which has become immobilized by adhesions becomes stretched out and distended to produce a small pouch superficially similar in appearance to that of a diverticulum. The contraction of the scar tissue at the site of fixation is of some importance, but more so is the mere fact that the esophagus is adherent at a given point.

Another plausible explanation for the formation of these pseudodiverticula is that of Kaufman, who maintains that each is the vestige of a healed or cicatrized perforation or fistula between the esophagus and a lymph node. That this is sometimes true is suggested by the fact that after the expectoration of a so-

called broncholith, representing the calcified remains of a peribronchial lymph node, a broncho-esophageal fistula may be left because of the fact that the node has eroded both the wall of the esophagus and that of the bronchus as a part of the same process (Fig 219) It should be pointed out however, that the tuberculosis is in the lymph nodes primarily and does not of itself involve the esophagus

It must not be assumed that tuberculosis is the universal cause of these abnormalities Any cause for acute lymphadenitis may be at fault Infection with pyogenic organisms, actinomycosis, anthracosis, and periesophageal abscesses may give rise to such a deformity

FIGURE 218 Drawing of a traction diverticulum showing the funnel shaped interior and the adhesions of the apex to inflamed lymph nodes of the sub carinal area (Esophagus opened along its right side close to the anterior surface and spread open toward the left) (After L. G  ry)



FISTULA
R BRONCHUS
ESOPHAGUS
AZYGOS V

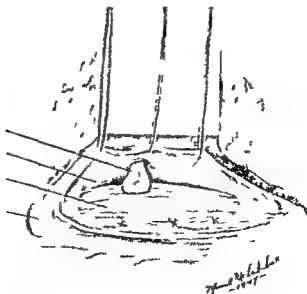


FIGURE 219 Drawing of traction diverticulum with a fistulous communication to the right main bronchus after the patient had expectorated several broncholiths representing calcified lymph nodes which had eroded the bronchial wall (Sketch made at operation)

Epibronchial pseudodiverticula never become large—rarely any larger than a walnut. They are usually attached to the anterior or right lateral wall of the esophagus, rarely to the left, and lie behind the bifurcation of the trachea. As might be anticipated, they may be multiple but always close to each other in the same general location. They are frequently irregular in shape. At the actual point of adherence the wall of the pouch is often nothing but scar tissue, which is further substantiation of Kaufman's view regarding their etiology.

Clinical Characteristics

The symptoms caused by these small niches in the esophageal wall may be vague or even nonexistent. Sometimes there is a slight degree of hesitation in the passage of food through the region where they lie, occasionally attended by pain which radiates to the back. Sometimes a patient will complain of regurgitation of food or liquid from that point, with a little substernal or subphoid pain followed by a feeling of relief as the ingested material finally passes down. This is undoubtedly the result of associated esophagospasm.

Not infrequently, however, these pseudodiverticula cause no symptoms whatever. In fact, if a patient with such a lesion complains of pain in the chest, another cause such as coronary arterial insufficiency or slow leakage from a ruptured aortic aneurysm should be sought before attributing the symptoms to the esophageal lesion alone.

Roentgen Examination

The roentgen examination is what establishes the diagnosis. Sometimes a mass representing the diseased adherent lymph nodes can be seen. The diverticulum may be seen best in the recumbent position with the pelvis elevated.



FIGURE 220 Traction diverticulum of the middle third of the esophagus. A Diverticulum as seen in the usual roentgen film partially filled with barium and air above. B tomogram showing better visualization of the diverticulum.

Sometimes it cannot be made to fill completely, when it may merely have the appearance of a superficial ulceration. Figure 220 shows a traction diverticulum of the midesophagus.

Esophagoscopy

Epibronchial pseudodiverticula of the traction type can rarely be discovered on attempts to visualize them through the esophagoscope. Often the only finding is a little bulging on the anterior wall of the esophagus and an appearance of fixation at that point.

Complications

Although they are usually asymptomatic they may give rise to complications, chiefly the lodgement of a foreign body at the site, perforation followed by the development of a periesophageal abscess, or eventually an esophago-bronchial fistula. Rarely an abscess may burst into the pleural cavity, the lung, the pericardium, or even into the vena cava or aorta with, in the latter case, a rapidly fatal outcome from hemorrhage.

Treatment

Usually no treatment is required. If mild symptoms of the nature described above arise, careful eating and the adoption of a bland, nonirritating type of diet are usually sufficient to give relief. If esophagospasm should develop atropine may be helpful. With an occasional exception, surgical excision is not indicated unless a fistula should develop between the esophagus and an adjacent bronchus, or rarely because of a periesophageal or pulmonary abscess. The preoperative preparation and choice of approach are the same as for epiphrenic diverticula. The incision in the right chest, however, should be made through the sixth intercostal space or bed of the sixth rib to provide better access to the subcarinal area where these diverticula develop.

Aftercare is the same as for removal of epiphrenic diverticula.

CHAPTER 12

Abnormalities of the Veins

BLEEDING is a frequent manifestation of certain diseases of the esophagus. From the prognostic point of view there are all degrees of seriousness, from a minute trace of blood with no clinical evidence of its occurrence to the catastrophic hemorrhage due to erosion of a major vessel ending in almost instantaneous death. Most of the time the bleeding is a symptom or a complication of an *intrinsic lesion of the esophagus itself*. Trauma may be the cause. Instrumental manipulation may provoke it and impacted foreign bodies which may perforate the wall of the esophagus and injure a neighboring vessel are occasionally responsible. Esophagitis, ulceration, or carcinoma may cause hemorrhage of varying degree. In other instances the esophagus serves merely as a channel by which the blood escapes from some other source such as an aortic aneurysm or a pulmonary vessel by way of a fistula into the respiratory tract.

Bleeding arising from esophageal vessels primarily, and not as a complication of any of the above disorders, is limited almost exclusively to three abnormalities as follows: (1) varices of the esophageal veins, (2) hemangiomata and (3) microscopic angiomatous areas observed in Rendu Osler disease. This chapter is devoted to these conditions only.

Esophageal Varices

There has been a renewal of interest in bleeding from esophageal varices because of the improvements in diagnosis and treatment which have been brought about in recent years. The first mention of this condition as a clinical entity was in a case report before the French Medical Society by LeDiberder in 1837 in which the enlargement of the spleen and the shrinking of the liver were commented upon. In the following year (1838) deFauvel in a second case report stressed the importance of alcoholic cirrhosis of the liver as a cause of the disorder. The etiological importance of this observation has gone unchallenged until relatively recent times and little interest was shown in the condition until late in the first quarter of the twentieth century.

The question arises as to whether the basic pathology in the development of varices involves an alteration in the structure of the vessel wall or whether it is simply dilatation of the veins. Most of the reports in the journals of pathological anatomy leave this point in doubt. Careful studies, however, have shown the importance and the constancy of lesions of the vein wall. For hemorrhage to occur, therefore, there must be something more than a purely mechanical factor. However that may be, the term esophageal varices is generally accepted even if it does not always have an actual anatomical significance.

Etiology and Pathogenesis

Although much is known about bleeding from esophageal varices, many points are still poorly understood. It is necessary to proceed from established facts to what is doubtful or unknown.

1 ABNORMALITIES OF THE PORTAL VEIN CIRCULATION. It is well recognized that the esophageal veins can serve as collateral circulation to relieve obstruction of the portal vein. The blood which cannot find its way through the liver into the inferior vena cava takes advantage of this anastomotic route among others to reach the heart by way of the superior vena cava and its affluents. This new circulation becomes established and usually develops progressively over a long period of time because there are few conditions which lead to a rapid occlusion of the portal circulation. Furthermore, the degree of development of the collateral circulation is proportional to the relative increase in pressure within the portal system.

The first disease of the liver which was invoked as a cause of this disturbance and the one which is still probably much the most frequent is *Laennec's atrophic cirrhosis*, often spoken of as alcoholic cirrhosis. The etiology of this form of cirrhosis has been the subject of much study in recent years and doubt has been cast upon the role of alcohol alone as the cause. Dietary deficiencies superimposed upon the effects of toxicity from infection, alcohol, or other poisons have been shown to play an important part in what is now recognized to be a complex mechanism.

The older concept of the clinical aspects of the disease was that the esophageal venous dilatations develop slowly and that their rupture is a late complication of the hepatic disease. It was thought always to occur at the same stage as that where bleeding from the dilated hemorrhoidal veins might be observed and long after the appearance of ascites and the periumbilical dilatations classically referred to as the *caput medusae*. Recent observations, however, cast doubt upon this sequence. It has been recognized that varices often develop in the absence of ascites. In fact this is a frequent occurrence but they may not be suspected until disclosed by the occurrence of hemorrhage. Actually, these venous dilatations may precede the ascites in point of time. This raises the question to be referred to later as to why this dilatation of the esophageal veins should proceed while there is still considerable blood going through the portal system even though ultimately the obstruction may become complete.

Another question which remains to be solved is why varices of the esophagus should develop in patients addicted to alcohol but who do not have cirrhosis of the liver. This might be explained, of course, by the development of a toxic

basis of extensive phlebitis and thrombosis of the portal vein. Furthermore, as proved at autopsy on patients who have died of hemorrhage from ruptured varices, the underlying cause of the portal obstruction may be cirrhosis of various types and etiologies other than that classically associated with the abuse of alcohol.

Other causes of portal vein obstruction are inflammatory processes secondary to cholecystitis and compression by tumors in the subhepatic region, inflammatory exudates, inflamed lymph nodes, or lymph node metastases from carcinoma at distant sites.

2 ABNORMALITIES OF THE SPLENIC CIRCULATION The next most frequent cause of esophageal venous dilatation is obstruction confined to the splenic vein. This condition was long misunderstood and is still under discussion today. Thrombosis of the vein is the usual occurrence which leads to the development of splenomegaly and the appearance of dilatation of the esophageal venous collateral circulation, the first manifestation of which may be the rupture of varices. The term *Banti's disease* no longer has the clinical significance it once had, at least from the etiological standpoint, but the syndrome is still observed. Thrombosis of the splenic vein is frequently a complication of acute or chronic pancreatitis, pancreatic abscess, or occlusion of the vein by invasion or compression by a tumor.

3 DISTURBANCE OF CIRCULATION IN THE SUPERIOR VENA CAVA Certain low cervical or superior mediastinal tumors may produce enough compression or invasive obliteration of the superior vena cava to cause venous engorgement of the esophageal veins along with the other veins involved by the back pressure. This situation is exceptional, and the seriousness of the esophageal venous dilatation is not as great as with hypertension in the portal circuit.

In rare instances no mechanical cause has been found. In these cases attempts have been made to invoke a predisposition of the veins of the esophagus in certain subjects to undergo dilatation and thinning of their walls. These changes eventually lead to rupture and hemorrhage. Certain authors stress the possibility of a congenital weakness of the veins as the basic difficulty in these obscure cases.

Although men predominate in every analysis of groups of patients, a fairly large number of women may also develop varices of the esophagus. The usual ratio is approximately two to one. The age of the patients varies. Bleeding from congenitally enlarged esophageal veins has been known to occur in infants. In infants and children also, certain diseases such as hereditary syphilis of the liver may give rise to varices of the esophagus. In adolescents and young adults, varices resulting from splenic vein thrombosis are the most frequent. In older patients obstruction of the portal system, usually intrahepatic due to cirrhosis of the liver is the usual cause.

Pathology

The immediate cause of the rupture of esophageal varices is variable. It may be a muscular effort such as violent coughing or straining acting upon a thin-walled varix. Sometimes it is trauma from the ingestion of coarse foods or the lodgement of a foreign body. Sometimes there is esophagitis over the dilated

veins Wangensteen has stressed the occurrence of superficial ulcerations of a peptic nature as the actual source of erosion and destruction of the vein wall resulting eventually in hemorrhage

The dilated veins may be confined to the lower esophagus but in the majority of instances the entire venous system at least in the thoracic portion of the organ, is affected to some extent. In the majority of instances also, the lower esophageal varices are continuous with dilatations of the gastric veins. Many times the periesophageal veins in the mediastinum are involved in enormous dilatations (Fig. 221)

At autopsies performed on patients who have died of hemorrhage from ruptured varices it is usual to find an ulceration over the dilated vein from which the bleeding came. Sometimes the defect in the vein may be as large as 2 to 3 mm. in diameter. Often it is much smaller. Exceptionally there may be multiple bleeding points. The rupture of the vein is undoubtedly caused by erosion from the exterior due to esophagitis and ulceration of the overlying mucous membrane. The element of stasis in the veins themselves is of course a contributing factor. Esophagospasm likewise tends to augment the difficulty.

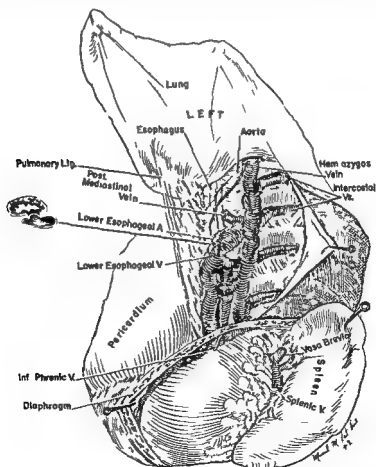


FIGURE 221. Drawing made at operation through a thoraco abdominal incision showing enormous dilatation of the periesophageal the subphrenic the intercostal and the hemi azygos veins (girl age 14 with splenic vein thrombosis and esophageal varices)

Histologic studies of autopsy material reveal periphlebitis and atrophic changes in the mucosa over the varices. Extravasation of blood into the cellular tissue around the veins may be found. Sometimes newly formed vessels filled with clots have been observed. Often thrombi in the process of resolution are seen. Submucosal extravasation of blood may be seen grossly as visible submucosal ecchymoses. Thrombosis of the dilated vessels can also be seen on gross inspection at autopsy. Sometimes the arteries may be involved as well as the veins, and sometimes both are involved at once.

Clinical Characteristics

Esophageal varices may be present for long periods of time before their existence is suspected as a result of a massive hemorrhage. In fact, bleeding evidenced by hematemesis or melena, or both, is their only manifestation. In the typical case the hematemesis from ruptured varices is characterized by the vomiting of bright blood which has not undergone any action by the gastric juice. It varies in quantity from small amounts to a veritable flood with rapid exsanguination and a fatal outcome. Sometimes with lesser hemorrhages the occurrence of bleeding is made known only by the passage of tarry stools. With massive bleeding the usual evidences of hemorrhagic shock consisting of sweating, pallor, tachycardia, thirst, and in severe cases syncope are observed.

Some patients experience premonitory symptoms before the onset of the attack. During several hours preceding the actual hemorrhage they may complain of malaise, vague discomfort, or a feeling of heaviness in the chest. Sometimes there is a little discomfort on deglutition, probably secondary to esophagospasm of variable duration and degree. Dysphagia is, however, rarely experienced.

Some patients, although they may not experience exsanguinating hemorrhages, may develop a state of chronic anemia from repeated small amounts of blood loss or continuous oozing. These patients will show a positive chemical test for blood in the stools. Some may remain chronically anemic for long periods before they succumb to a final massive hemorrhage. Others may have single episodic hemorrhages which stop short of exsanguination, leaving the patient in a state of weakness and anemia.

Roentgen Examination

Although the impression is abroad that the roentgen diagnosis of esophageal varices is easily accomplished, the fact is that this is not always the case, especially when the dilatations are small. In some difficult cases, therefore, it is wise to combine the roentgen examination with an esophagoscopy. Many observers recommend using a thick barium mixture. The examination is performed with the patient in the erect position followed by a shift to the left lateral decubitus to slow down the passage of the material through the lower esophagus where most of the varices are found. Sometimes it is necessary to make attempts to produce a degree of stagnation of the blood in the venous system of the esophagus in order to make the varices swell and assume their maximum size. The use of the Trendelenburg position may be helpful for this purpose or, even better, the Valsalva maneuver where the patient is made to

attempt a forced expiration with the glottis closed. This increases the intrathoracic pressure and, with the additional squeezing of the contracted diaphragm, distends the periesophageal veins to produce temporary stasis in the whole venous network (Fig. 222, A, B).

Three degrees of enlargement of the veins can be distinguished. In the first phase there is a slight diffuse venous congestion. The roentgen film discloses merely a pattern of somewhat enlarged mucosal folds in the lower third of the esophagus. With a small amount of barium the major fold particularly appears enlarged and irregular, although its normal contour is preserved. In this stage it is difficult to establish a definite diagnosis by means of roentgen examination alone.

In the second phase of development the most important veins are dilated. Here faint lacunary shadows appear in the lower esophagus. Just above the sphincter-like epicardial segment a slight accentuation of the mucosal relief can be seen. This gives an appearance reminiscent of pseudopolyposis. The shape of these shadows, however, varies during the course of the same examination depending upon the degree of filling of the veins.

In the third or fully developed phase the enlarged veins bulge visibly from the mucosal surface and give the typical appearance described by Wolf, Schatzki, and others. The mucosal relief is very irregular. It is replaced by longitudinal furrows or by large longitudinal hollows which are either regular or sometimes irregular or sinuous in shape. They often present the appearance of a group of alveoli or of a section of honeycomb or of a clover leaf. The contours of the esophagus are irregularly draped with semilunar-shaped prominences (Fig. 223) giving it a worm-eaten appearance. The epicardial segment, because of its normal tonicity, is free from these changes. If associated gastric varices are pres-

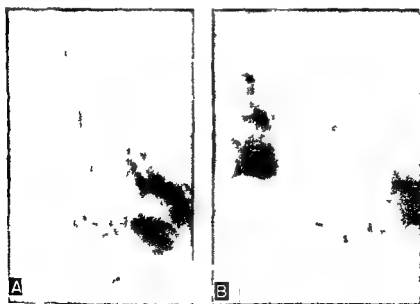


FIGURE 222 Roentgen films of a patient with esophageal varices. A During quiet normal respiration. B during the Valsalva maneuver.

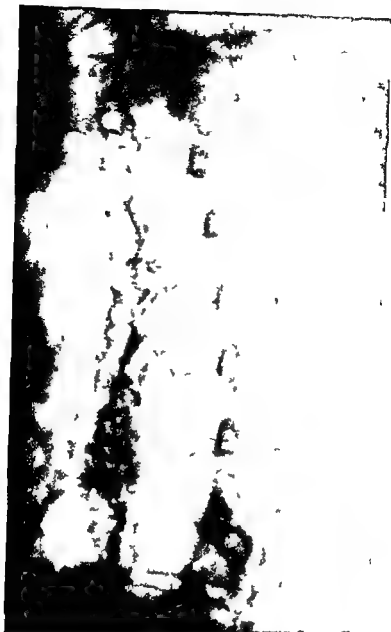


FIGURE 223 Roentgen film of esophagus showing varices extending almost the entire length (Israelski)

ent, the appearance is that of pseudopolyposis both above and below the diaphragm

The flexibility of the esophageal wall is maintained. The esophagus undergoes a normal degree of distention when a large mouthful of barium is swallowed. This makes it possible to eliminate from the diagnosis any infiltrating lesion whether inflammatory or neoplastic.

The transit of material through the esophagus is somewhat slow if the varices are unusually large. Even when peristalsis is present, it is more leisurely than usual. The shadows disappear when a peristaltic wave passes by.

As would be expected, the difficulty of diagnosis is augmented by the presence of a complicating ulcer or esophagitis.

The difficulty of roentgen diagnosis of esophageal varices is demonstrated by the occasional occurrence of fatal hemorrhages from dilated veins not discovered at roentgen examination a short time before

Esophagoscopy

Many times the varices can be demonstrated at esophagoscopic examination. They can be seen as venous cords beneath an inflamed mucous membrane (Plate III, 2). Although this appearance is sufficiently characteristic to establish a diagnosis, it is probably best to refrain from making an esophagoscopic examination because of the danger of injuring the thin, fragile mucosal layer over the dilated vessels. Major hemorrhage might result from minor trauma induced in this way. In spite of this, esophagoscopy is utilized by some clinicians to differentiate between hematemesis from varices of the esophagus and that which comes from the stomach.

Differential Diagnosis

On the basis of the roentgen appearances, varices may in rare instances be mistaken for a polypoid carcinoma. The clinical history in carcinoma is so obviously different from the relative lack of symptoms in varices, other than bleeding, that no such mistake should be made.

When confronted by a patient with massive hematemesis, one may experience great difficulty in distinguishing the cause. The only observation which might suggest that the bleeding is from esophageal varices is the fact that the blood is bright and alkaline, whereas the blood which is vomited from the stomach is darker, often with coffee ground material in it, and is acid in reaction. This differentiation, however, is not conclusive because blood from bleeding varices may flow into the stomach first, where it may remain for variable periods of time before it is vomited.

With carcinoma which rarely bleeds massively, with benign tumors which are unusual and which almost never cause hemorrhage and with ulcers from esophagitis which may bleed massively, the clinical history is usually conclusive in excluding varices as the cause.

With patients who are known to have cirrhosis or in those who have had a diagnosis of varices made previously by roentgen examination, the presumption is that the esophageal varices are the source of the bleeding. Even then the diagnosis may not be correct because, in approximately 12 per cent of patients with known varices who develop massive upper gastrointestinal tract bleeding, the hemorrhage comes from a duodenal ulcer and not from the esophageal veins. The most difficult diagnostic problem occurs in this group and in those who have no obvious disease.

It is in these two groups of patients that great assistance is obtained from a specially devised tube originated by Sengstaken and Blakemore (Fig. 224). This tube is provided with two rubber balloons, one of which is near the end and the other, a longer one, is attached a short distance proximal to it. The tube is inserted into the stomach and the distal balloon is distended by injecting air through the corresponding lumen. The tube is then pulled back so that the

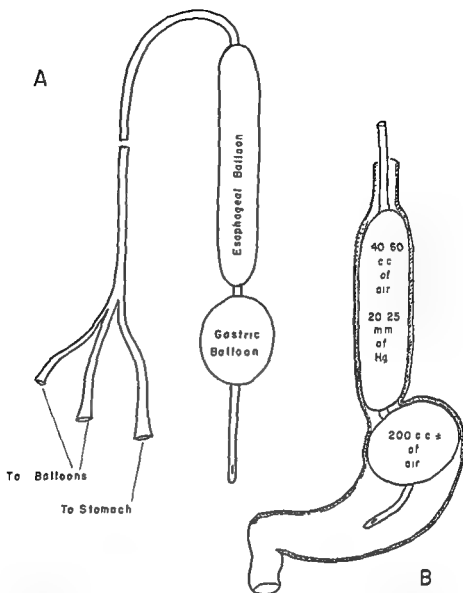


FIGURE 224 Diagram of the Sengstaken Blakemore tube *A* Tube showing balloons and the proximal ends of the three lumina *B* the tube in correct position for diagnosis and treatment

distended intragastric balloon is held tightly against the cardia. The upper or esophageal balloon is next distended through its connecting lumen. The effect of this is to compress the mucosa of the lower third of the esophagus from which the bleeding in esophageal varices usually occurs. The third lumen, which connects freely with the stomach, is then used to lavage the stomach until the washings are clear. This lumen is then placed on suction. Observation of the patient over the next few hours will usually demonstrate whether the bleeding comes from the stomach or from the esophagus. If the tube has been inserted and adjusted properly, the bleeding will stop immediately if it comes from the esophagus. If the fluid aspirated from the stomach continues to be bloody, however, it can be assumed that the bleeding must be coming not from the esophagus but from the stomach or duodenum. The value of this technique both as a method for diagnosis and as a means of stopping the hemorrhage is obvious.

Prognosis

The prognosis of bleeding from esophageal varices is always grave. Approximately four-fifths of the patients die if unsuccessfully treated. Nowadays the prognosis is better if the condition is recognized as a result of roentgen examination in a latent period before bleeding has occurred. The patient can then be kept under careful observation and surgical intervention undertaken as soon as the need becomes obvious.

Treatment

The treatment of esophageal varices has been the subject of much interest in recent years, particularly since the development of measures to reduce the pressure in the obstructed portal system.

If varices are discovered before hemorrhage has occurred, the question arises as to whether one should attempt to obliterate them or reduce the pressure within them in an effort to prevent serious consequences. Opinions vary, but the condition of the patient is an important factor in the decision. In patients with cirrhosis of the liver who have not experienced hematemesis of alarming proportions, every effort should be made to improve the condition of the liver. It is surprising how much can be accomplished by a regimen of high protein and high carbohydrate intake, vitamin B complex administration, omission of fat from the diet, and the withholding of alcohol. The ascitic fluid is often absorbed, the patient's color and appearance improve, the liver function tests tend to revert to normal, and in many instances the patient can be rehabilitated to a large degree. The condition of the varices, however, is irreversible by any medical means, and the roentgen appearance remains unchanged. Those who have experienced bleeding furthermore do not gain any appreciable degree of freedom from attacks.

Measures which have been devised to prevent the serious consequences of hemorrhage comprise (1) those directed toward eliminating the veins by a direct attack upon them and (2) those which by indirect means are intended to reduce the intravenous pressure to the point where life-endangering hemorrhages are less likely to occur.

DIRECT MEASURES The measure first devised to obliterate the varices was the injection of a sclerosing solution into the dilated veins through the esophagoscope. This has been given extensive clinical trial in various parts of the world. Several sclerosing agents have been tried, notably quinine and urethan hydrochloride and sodium morrhuate. The technique is difficult because of the necessity for working at a great distance through a narrow instrument. Furthermore, as with the injection treatment of varicose veins of the legs, the results have been disappointing because the treatment is not directed toward overcoming the underlying cause, namely the hypertension in the portal venous system. Temporary ablation of the veins can be accomplished but the rate of recurrence is high. The method should be reserved for those patients whose condition does not permit a more effective surgical approach to the problem.

The second measure directed against the dilated veins themselves consists in suturing the varicosities. This is accomplished through a left thoracotomy incision. The lower esophagus is opened longitudinally and the varicose veins

are sutured over and over, each with a continuous suture of chromicized catgut. Usually two or three masses of veins requiring obliteration by suture are found. The esophagotomy incision should be closed lengthwise with two layers of 5-0 interrupted silk sutures. Under no circumstances should closure be made in a circumferential direction as would be done if the opening were in a portion of the intestine. The esophagus does not elongate readily to accommodate this type of closure without too much tension. Furthermore, its ability to dilate circumferentially permits the use of a lengthwise closure without fear of stenosis. Fatalities have been observed as a result of failing to observe this precaution.

Unfortunately this method of eliminating esophageal varices, though more quickly accomplished, is no more effective in bringing about a permanent occlusion of the veins than is the injection of sclerosing agents. Its use, therefore, should be confined to stopping the bleeding from varices which cannot be controlled by means of esophageal balloon tamponade. The surgeon, however, must be prepared in every such patient to proceed with a portacaval or splenorenal anastomosis within a few weeks of the performance of the emergency ligation of the varices. At the Massachusetts General Hospital where this measure has been employed in a large series of patients as an emergency measure, recurrent hematemesis has been observed in the majority of the patients within three months after the ligation was performed unless a vascular shunt was performed to relieve the portal hypertension.

As a third means of direct attack upon the varicose veins themselves in an effort to control severe bleeding, *balloon tamponade* has achieved considerable popularity. There is no doubt that this measure has saved many patients from death by rapid exsanguination. Unfortunately the bleeding tends to recur after the pressure is released. The use of this technique should be confined, therefore, to the temporary control of massive bleeding while the patient is being prepared for an operation either to ligate the veins or, better, to create a decompressing vascular anastomosis. The triple lumen, double balloon apparatus is preferred because it provides in addition a means of making a differential diagnosis as to the source of the hemorrhage as described on page 283.

The tube is introduced through the nose to the point where the gastric balloon is assumed to be in the stomach. This balloon is then distended with air injected through the corresponding lumen and the tube is pulled back firmly against the cardia. This maneuver serves the double purpose of securing the correct alignment of the esophageal balloon and of compressing what dilated veins there may be in the circumcardial region of the stomach. For this purpose the gastric balloon is made unusually large with a capacity of 300 to 400 cc., although it is rarely necessary to inflate it to its fullest extent. In the average case approximately 200 cc. of air in the gastric balloon will suffice. The esophageal balloon is distended with the amount of air which is just sufficient to compress the varices against the underlying muscular wall of the esophagus. The amount of pressure required for this purpose is in the vicinity of 20 to 25 mm. of mercury. To accomplish this it is necessary usually to inject 40 to 60 cc. of air (Fig. 224).

After the hemorrhage has been brought under control, the patient may be

fed liquid nourishment through the lumen, the end of which lies free within the stomach

INDIRECT MEASURES The greatest hope for patients who have bleeding esophageal varices springs from the performance of an operation which creates a by pass or shunting of the blood from the portal system where the elevated pressure is a constant menace threatening the rupture of the varices, to the systemic venous system by way of the vena cava where the pressure is low. That this procedure is effective is borne out not only by the safeguarding of the patient against the occurrence of subsequent hemorrhages in over 60 per cent of the cases but also by the demonstration of a striking fall of the pressure in the dilated veins of the obstructed portal system by direct measurements made at operation. A fall in pressure from the vicinity of above 300 mm. of water to below 200 mm. of water is not unusual. Two methods are available.

Splenorenal Anastomosis The first is the performance of an anastomosis between the splenic vein after splenectomy and the left renal vein. This procedure was introduced in 1945 by Blakemore and Lord who developed an ingenious method of making the anastomosis by using a vitallium tube. This technique of anastomosis has since then been abandoned in favor of the suture method. The creation of a splenorenal anastomosis is particularly desirable in the patients with extrahepatic portal obstruction especially those with splenic vein or portal vein thrombosis. On the other hand certain surgeons prefer this procedure over the portacaval shunt even in intrahepatic obstruction due to cirrhosis because of the obvious disadvantages of creating an Eck fistula. The chief advantage of the splenorenal technique therefore is that it avoids a complete by passing of the liver.

The disadvantages are, first, that it may be impossible to find a suitable vessel to use. As a result of previous thrombosis the splenic vein is sometimes obliterated or replaced by a network of canaliculi. When this is the case, the operation must be abandoned. Some benefit may result in such a patient from the removal of the spleen but recurrences of hemorrhage are frequent. In this group of patients therefore, if they do not succumb to a massive hemorrhage in the interval, recourse must be had to the performance of a portacaval anastomosis at another time. A second disadvantage is that the operation is considerably more difficult even with favorable vessels although this should not in itself be used as a reason for choosing the more easily performed portacaval technique. Because it is less time-consuming therefore, it may be wise to choose the latter method in the sickest patients. The third objection to the method is that because of the relatively small size of the splenic vein in many instances, the volume of blood redirected through the shunt may not be adequate. For the same reason no doubt, there is also a tendency of the shunt to fail to remain open because of thrombosis at the site of the anastomosis. For these reasons, some surgeons now advocate the use of a portacaval anastomosis as the primary procedure.

Portacaval Anastomosis The second method of relieving hypertension in the portal venous system is the performance of a portacaval anastomosis. This may be done either end to side or side to side between the dilated portal vein and the inferior vena cava. The tendency is present is to interrupt the portal

vein and make the anastomosis end to side in the belief that the shunt is more likely to remain open and is mechanically more effective. Although this method is more easily accomplished in most instances than a splenorenal anastomosis, difficulties are sometimes encountered with it as well. The greatest technical problem arises when the portal vein is found obliterated because of thrombosis. In this event it is sometimes possible to bring about a degree of relief in a very roundabout way by dividing the inferior mesenteric vein near its junction with the splenic vein for the performance of a shunt end to side with the vena cava.

The technical advantages of using a portacaval shunt to overcome portal hypertension as a means of preventing hemorrhage from esophageal varices have been stressed. It is still not possible to say, however, how serious the physiological effects of the complete by-passing of the portal blood around the liver may be upon the individual. Many surgeons, therefore, believe that until more time has elapsed for the further study of patients who have been subjected to the performance of what is actually an Eck fistula, it is preferable to use a splenorenal anastomosis if at all feasible.

TECHNICAL CONSIDERATIONS—The choice of anesthesia for patients with hepatic damage presents a problem. The most important consideration is avoidance of hypoxia. In fact, the most obvious cause of hepatic coma, other than ammonia intoxication from the absorption of the products of decomposed blood in the intestinal tract, is hypoxia occurring during the performance of the operation or during the recovery period.

Another consideration is the technical problem presented by the excessive bleeding from innumerable dilated small veins which must be controlled in order to prevent excessive losses of blood. A related complication of this is the fact that following the administration of several pints of citrated blood to make up for the blood lost during the operation the amount of citrate reaches the point where the clotting mechanism is interfered with. The bleeding thereafter tends to be more difficult to control and a vicious circle is set up. A shift to the administration of freshly drawn blood after a certain amount of previously stored bank blood has been given is very helpful.

A more effective way to get around this difficulty, however, is to reduce the amount of blood loss and the need for transfusions by lowering the patient's systemic blood pressure during the course of the operation. This may be accomplished by chemical means, but usually advocated is the administration of hypotensive spinal anesthesia. The anesthetic agent is injected continuously and slowly during the operation through a small caliber polyethylene catheter introduced into the spinal canal through a lumbar puncture needle. The patient is kept asleep and the expansion of the lungs is controlled by the administration of nitrous oxide and oxygen through an endotracheal tube. By the use of this technique the operating time can be greatly reduced because of the marked diminution in the amount of bleeding from the smaller vessels. This shortening of the procedure alone improves the prospects of recovery by an appreciable degree.

Technique of Splenorenal Anastomosis—The patient should be placed squarely on his right side. The incision starts over the left rectus sheath about

midway between the umbilicus and the xiphoid process and extends transversely to the costal margin where it turns to follow the direction of the tenth intercostal space beyond the angle of the rib. The intercostal space is opened all the way posteriorly and the diaphragm is incised part way in the direction of the central tendon. After the wound edges have been protected with gauze pads and the rib spreader adjusted, a wide exposure of the left upper quadrant of the abdomen is obtained. The lateral peritoneal reflection from the hilus of the spleen is divided and the spleen and tail of the pancreas are turned up to expose the splenic vein from behind. The gastrosplenic ligament is divided along with the vasa brevia and the left gastroepiploic artery and vein. The splenic flexure of the colon is reflected downward. These steps make it possible to move the spleen upon its vascular pedicle in order to dissect the vein from its bed in the pancreas. To accomplish the freeing of the vein a number of small fragile pancreatic branches must be tied and divided. After a sufficient length of vein has been freed the splenic artery is ligated and severed. An occlusive clamp is placed across the splenic vein close to the point where it emerges from the pancreas and the spleen is removed after cutting across the vein at its first branching in the splenic hilus.

The next step consists in the exposure of the left renal vein. This is readily accomplished by reflecting the perirenal fat. Enough of the renal vein is freed by dissection to make it possible to put a curved blood vessel clamp on its anterior portion in a tangential direction so as to isolate a portion of the anterior wall for the performance of the anastomosis. In this way the necessity for temporarily occluding the renal circulation is avoided. A linear incision of appropriate length is made in the renal vein and after both veins have been washed with a solution of heparin, an end-to-side anastomosis is made using a continuous suture of 5-0 or 6-0 silk on an atraumatic needle. The approximation is made without eversion of the edges. Upon the release of the clamps the blood can be seen rushing through the anastomosis from the splenic vein where the pressure is high to the renal vein where the pressure is much lower.

The incision is closed in layers with interrupted sutures of silk. The ribs are held in position with pericostal or percostal sutures of No. 1 chromic catgut. It is wise to leave a Foley catheter in the lower portion of the left pleural cavity for postoperative closed suction drainage.

Technique of Portacaval Anastomosis. The patient is placed on his left side and an abdominothoracic incision is made with extension into the tenth intercostal space in exactly the same manner as that used on the left for a splenorenal anastomosis. The liver is retracted upward and the vena cava and portal vein are exposed to view. An occlusive blood vessel clamp is placed on the portal vein several centimeters from the porta hepatis. The vein is then divided as close as possible to the liver. The hepatic end is ligated or sutured over with silk. A clamp of the Satinsky type is placed tangentially on the inferior vena cava to isolate a portion of the vessel for the anastomosis while permitting the flow of blood to continue through the remainder of the vessel. Using a fine atraumatic silk suture an end-to-side anastomosis is made with simple apposition of the vessel edges exactly like that employed for a splenorenal anastomosis.

A lateral anastomosis may be performed if preferred. The incision is closed in the same manner as that employed on the left for the splenorenal shunt.

AFTERCARE : With the exception of special measures which may be required to support the liver in patients who have cirrhosis, the aftercare does not differ from that following any thoracotomy. The details are not a subject for consideration here.

RESULTS When used for the control and prevention of hemorrhage from esophageal varices, either procedure usually gives good results. Prevention of subsequent attacks of massive bleeding occurs in approximately 60 per cent of the patients. The mortality with improved preparation of the patient and the use of modern anesthetic techniques should not exceed 10 to 12 per cent of the patients. The long-term results are not yet clearly established. Many patients die within the ensuing months or years of hepatic insufficiency due to a progression of the underlying disease of the liver. Certain patients who have had a splenorenal anastomosis will bleed again after the shunt has presumably become occluded by thrombosis. Some of these patients are relieved once more by reoperation, this time for the performance of a portacaval anastomosis. Others may succumb from hemorrhage or hepatic disease before another operation can be performed. Several patients have been observed in whom subsequent fatal hemorrhages occurred even though the shunt remained patent as proved by postmortem examination.

Although the performance of a venous shunt is palliative rather than curative in terms of an attack upon the basic disease, great benefit has resulted for a large number of patients from its use. It is the best method yet devised to deal with the problem of bleeding esophageal varices. It should be pointed out, however, that in the majority of instances the veins can still be seen on roentgen examination even though the pressure within them has been reduced to a level where hemorrhage is unlikely to occur.

OBSELETE SURGICAL PROCEDURES Before the development of the shunting procedures attempts were made to relieve the pressure in the varices by various other less effective or completely ineffective measures which should be mentioned. A few patients were prevented from experiencing further hemorrhages by simple ligation and division of the splenic artery. This was most likely to be effective in the rather unusual instance when the bleeding came from gastric varices in the upper half of the stomach and not from the esophagus. A few patients also, were relieved by splenectomy but the results of this operation were by no means satisfactory. Recurrence of bleeding took place in the majority of instances.

Other methods included ligation of the hepatic artery, ligation of the gastric veins, ligation of the left gastric and left gastroepiploic arteries leaving the veins intact, ligation of the periesophageal veins, transection of the stomach and division of the left gastric vessels and vasa brevia with reapproximation of the divided edges (Tanner), and total gastrectomy as advocated by Wangensteen to remove any possibility of acid peptic erosion of the mucosa over the veins. All of these procedures have been abandoned either because they were shown to be ineffective or because they were too drastic.

Hemangiomas of the Esophagus

These lesions should actually be regarded as benign tumors. They are so rare that they do not have much clinical importance. They have been observed both in children and in adults. Chevalier Jackson has observed several cases and advises removal with a biopsy forceps. This he has accomplished without serious bleeding. Guisez observed a case of an angioma 15 to 20 mm. in diameter, rather flat and elongated in shape, and located 2 cm. above the cardia. The tumor disappeared completely after radium therapy.

Familial Hemorrhagic Hemangiomatosis (Rendu-Osler Disease)

This rare disease, characterized by the occurrence of multiple small hemangiomas or telangiectases of the gastrointestinal tract, must be mentioned as an unusual but possible source of hematemesis. Whether or not the bleeding in this disease is ever confined entirely to lesions in the esophagus itself is not known.

Acute and Chronic Nonspecific Esophagitis

Esophagitis

Strictly speaking, the term *esophagitis* should be used to designate any inflammatory process involving the esophagus. It is not always distinguished as a disease entity of itself although, as Chevalier Jackson points out, it is more common than usually supposed. Reports based upon routine autopsy examinations reveal the presence of esophagitis in approximately 7 per cent of the subjects. As a result of careful histological examination of the esophagus in 100 autopsies, Schubert found only thirty-seven with no evidence of involvement. These results, however, do not agree with clinical statistics, probably because the autopsy reports are based upon the examination of patients who had died after long, wasting illnesses or after surgical interventions. Actually the clinical importance of the condition should not be exaggerated, particularly when it comes to the insistence upon a diagnosis of esophagitis to explain certain symptoms commonly thought of as characteristic but which may be due to esophagospasm or other causes.

The subject of this chapter is simple, uncomplicated esophagitis. Phlegmonous esophagitis, that resulting from chemical burning, that due to allergic causes, specific infections, and other forms will be discussed in subsequent chapters.

Nonspecific esophagitis, though usually acute and self-limited, may evolve into a chronic form which is more common because it accompanies many other disorders of the digestive tract. It is often recognized in the course of endoscopic examinations performed for the study of other conditions.

Acute Esophagitis ("Catarrhal")

Acute nonspecific inflammation of the esophagus may accompany acute upper respiratory infections of a grippelike nature acute tonsillitis, acute pharyngitis, acute laryngitis, pneumonia, and especially the eruptive diseases like measles and scarlet fever. The esophagitis complicating many of these diseases develops by continuity, descending from the inflamed area above. The proof of this is supported by anatomical sections and the finding of the causative or complicating organism, often the streptococcus in the exudates and in the submucosal tissues of the pharynx and esophagus, chiefly in children. Infection of the esophagus by way of the blood stream may explain the exceptional occurrence of esophagitis in pneumonia peritonitis, or pyelonephritis.

On the other hand, the esophageal epithelium is susceptible to the irritating effects of gastric juices resulting from regurgitation or vomiting in patients with hyperchlorhydria associated with obstruction of the pylorus or duodenum. This may be thought of as ascending esophagitis but unlike the descending esophagitis mentioned above it is not primarily bacterial in origin. This phenomenon explains in part the frequency of inflammation in the lower third of the organ. It usually means also that the protective film formed by the mucus from the esophageal glands is deficient. Once an inflammatory process has developed the obstruction of the mucous glands prevents the replenishment of the film of mucus and favors further injury to the surface. The condition may occur also as a complication of acute gastritis.

Acute esophagitis following the administration of an inhalation anesthetic results probably from the combined vasodilating effect of the anesthetic agent and the local irritation produced by postoperative vomiting. Other local causes are the prolonged use of an intubating intranasal tube (Levin, Miller-Abbott, etc.) the abuse of bougienage, repeated lavage of the stomach, more rarely the use of intraluminal radium applicators, and the effects of roentgen therapy.

From the *histopathologic point of view* two forms can be distinguished.

1 The first is the desquamative type characterized by a partial separation of the esophageal epithelium which is replaced by diffuse leucocytic infiltration (Fig. 225, c). The examination of the desquamated mucosa shows that the lesions are similar to those of hereditary bullous epidermolysis. Both conditions manifest the same clinical course and rapid healing.

2 The second is acute or subacute exudative esophagitis characterized by a massive diffuse infiltration of the esophageal epithelium with various inflammatory elements (Fig. 226). The disturbance may involve the submucosa also. This type demonstrates all the phases of an acute inflammation.

Ulceration of the mucosa is a rare occurrence except in unusual instances particularly in certain acute infections like diphtheria or in some cases of septicemia. Actually the ulceration in these cases is superficial and results from limited areas of necrosis. Healing is rapid. This condition is not to be confused with so-called peptic ulceration of the esophagus.

CLINICAL CHARACTERISTICS The only symptom is usually a slight pain referred to the cervical region, behind the sternum between the shoulder blades or in the epigastrium depending on the segment which is involved. The



FIGURE 225 Photomicrograph Subacute desquamative esophagitis ($\times 105$) a, Malpighian stratified pavement type epithelium, b, area which is denuded of epithelium c, focus of leucocytic infiltration d, abnormally dilated blood capillary of the chorion e, longitudinal smooth muscle fibers (H. L. Gubert)



FIGURE 226 Photomicrograph Subacute exudative esophagitis ($\times 125$) a Epithelial lining infiltrated with leucocytes at b and c along with red blood cells which are most marked where they infiltrate the chorion at d e bundles of smooth muscle fibers of the muscularis mucosae seen in cross section

saliva may be swallowed with a certain amount of difficulty and the patient may experience delay in the passage of food down the esophagus

ESOPHAGOSCOPIC EXAMINATION reveals intense redness of the mucosa with marked injection of the blood vessels and often an overlying film due to the

exfoliation of the epithelium. The swelling of the mucosa is rarely sufficiently pronounced to impede the passage of the esophagoscope.

TREATMENT The treatment of acute esophagitis consists in the restriction of intake to bland liquids and the exhibition of subcarbonate of bismuth or Gelusil to protect and soothe the irritated mucous membrane. Complete healing usually occurs in several days.

Agranulocytic Esophagitis

Localization of agranulocytosis in the esophagus is a rare occurrence. The first reported case was that of W. Schultz in 1929. In the same year E. Reyne reported a series of 18 patients with agranulocytosis, two of whom at autopsy showed esophageal involvement along with that of the pharynx, larynx, lips, stomach, and intestine. Others were reported by Pallesstrini and Chevalier Jackson. These, however, were all postmortem observations. In the majority there was extensive involvement of the lower two-thirds of the organ with a pseudomembranous esophagitis. As with all forms of agranulocytosis, there seems to be a predominance in women over fifty years of age.

One of the few patients with this disease who survived is a woman, the roentgen film of whose esophagus is shown in Figure 227. She had been given a long course of sulfathiazole because of a chronic pyelitis. When she reported to her physician with a fever, sore throat, and a membranous exudate over the surface of the pharynx, she was given more tablets to take on the mistaken as-



FIGURE 227. Esophagogram of a patient with stricture of the esophagus resulting from agranulocytic esophagitis.

sumption that the pharyngitis was bacterial in origin. The second drug administered was sulfadiazine, 1 gram every three hours. The patient took this medication for eight days when she rather suddenly developed hyperpyrexia, became delirious and then comatose. She was found to have a white blood cell count of 1200. After a period of heroic treatment she began to recover, but her first realization as she awoke from her comatose state was that she was unable to swallow without pain and obstruction. After approximately three weeks the dysphagia became so pronounced that she could swallow only thin liquids. A partial esophagectomy with a supra-aortic arch anastomosis was performed. The pathological examination of the esophagus showed marked constriction with thickening of the esophageal wall to 8 or 9 mm. The mucosa appeared hemorrhagic and in the strictured area it was completely denuded. The microscopic examination showed evidences of acute and chronic inflammation with ulceration of the mucosa. Few such cases have been recognized in the living and the treatment remains symptomatic and empirical following the elimination of any recognizable causative agent.

Chronic Esophagitis

Chronic esophagitis, although its symptomatology is less striking than the acute form, is nevertheless better understood because it is a rather frequent finding in esophagoscopy examinations or because it is encountered at autopsy in patients afflicted with a chronic disease of the digestive tract or the circulatory system, or in those who have died after surgical intervention upon the abdomen.

Various causes have been described. The first has to do with food and liquids ingested, particularly the excessive use of strong alcoholic drinks or highly spiced foods or the ingestion of liquids which are too cold or above all too hot. Excessive smoking and especially chewing of tobacco are sometimes incriminated. These faults of hygiene give rise most often to the hyperemic or catarrhal form of esophagitis, spoken of as *exfoliative*.

Minor thermal injuries to the mucosa provoke an epithelial proliferation accompanied by edema and finally a degeneration of the epithelial layer. Less frequently one sees papillary hyperplasia or leucoplakia.

The second factor is the effect upon the mucosa of stasis of food material, especially in chronic stenosis and proximal dilatation of the organ. Chronic esophagitis is an almost constant finding in cicatricial stenosis of the esophagus and a frequent one in mega-esophagus.

A third cause of chronic esophagitis is the congestion due to chronic venous stasis resulting from cardiac decompensation, pulmonary sepsis, or particularly portal hypertension from cirrhosis of the liver or thrombosis of the splenic vein.

There may also be secondary or accessory causes such as occupational hazards notably the ingestion of fine particles of metal or porcelain by metal polishers or porcelain workers, and chemical effects such as that resulting from regurgitation of highly acid gastric juices. In the latter case many times there is undoubtedly a constitutional predisposition (Chapter 15).

In some instances local trauma, particularly the effect of pressure by an intubing nasogastric tube, may be a contributing cause.

Pathological Anatomy

Chronic esophagitis, like any chronic inflammatory process elsewhere in the body, may appear as an acute or subacute process arising from irritative disturbances of more or less prolonged duration. These may be the action of physical agents or chemical irritants, bacterial invasion as with syphilis or tuberculosis, or involvement by various fungus infections like actinomycosis. As with acute esophagitis, purely inflammatory changes predominate but chronic inflammation, especially when there is venous stasis, becomes complicated by sclerosis which is a frequent sequel. This is characterized by thickening of the epithelium, polypoid or papillary hyperplasia, lymphocytic infiltration and hypertrophy of the submucosal, muscular, and glandular layers, venous hyperemia, and interstitial hemorrhage throughout (Fig. 226).

These changes predominate in the areas which are particularly subject to irritation. They may become complicated by the occurrence of acute exacerbations and may lead to ulceration or even areas of necrosis.

These manifestations are characteristic of exfoliative esophagitis as described by Reichmann and by Patterson. Under the microscope it is obvious that several elements of the esophageal wall are involved at once. In the mucous membrane layer the most characteristic change in long-standing chronic esophagitis is leucoplakia. It is well known that leucoplakia is represented in dermo-papillary mucous membrane layers by the abnormal presence of a granular infiltration with abundant hyaline changes diffused throughout between the Malpighian bodies and the extremely thickened cornified layer (Fig. 228).

Certain authors have considered this particular condition to be a transitional phase from chronic inflammation to cancer as though the leucoplakic process

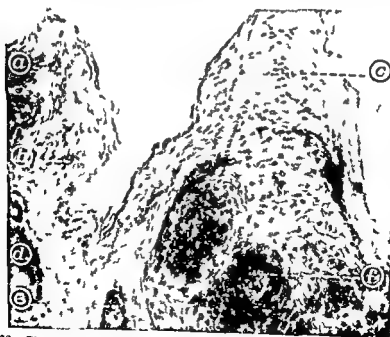


FIGURE 228 Photomicrograph Leucoplakia of the esophagus ($\times 180$) granules hyaline and eleidine at *a b* and *c* papillomatosis at *d e* and *f* (H. L. Gui)

sumption that the pharyngitis was bacterial in origin. The second drug administered was sulfadiazine, 1 gram every three hours. The patient took this medication for eight days when she rather suddenly developed hyperpyrexia, became delirious and then comatose. She was found to have a white blood cell count of 1200. After a period of heroic treatment she began to recover, but her first realization as she woke from her comatose state was that she was unable to swallow without pain and obstruction. After approximately three weeks the dysphagia became so pronounced that she could swallow only thin liquids. A partial esophagectomy with a supra-aortic arch anastomosis was performed. The pathological examination of the esophagus showed marked constriction with thickening of the esophageal wall to 8 or 9 mm. The mucosa appeared hemorrhagic and in the strictured area it was completely denuded. The microscopic examination showed evidences of acute and chronic inflammation with ulceration of the mucosa. Few such cases have been recognized in the living and the treatment remains symptomatic and empirical following the elimination of any recognizable causative agent.

Chronic Esophagitis

Chronic esophagitis, although its symptomatology is less striking than the acute form, is nevertheless better understood because it is a rather frequent finding in esophagoscopy examinations or because it is encountered at autopsy in patients afflicted with a chronic disease of the digestive tract or the circulatory system, or in those who have died after surgical intervention upon the abdomen.

Various causes have been described. The first has to do with food and liquids ingested, particularly the excessive use of strong alcoholic drinks or highly spiced foods or the ingestion of liquids which are too cold or above all too hot. Excessive smoking and especially chewing of tobacco are sometimes incriminated. These faults of hygiene give rise most often to the hyperemic or catarrhal form of esophagitis, spoken of as *exfoliative*.

Minor thermal injuries to the mucosa provoke an epithelial proliferation accompanied by edema and finally a degeneration of the epithelial layer. Less frequently one sees papillary hyperplasia or leucoplakia.

The second factor is the effect upon the mucosa of stasis of food material, especially in chronic stenosis and proximal dilatation of the organ. Chronic esophagitis is an almost constant finding in cicatricial stenosis of the esophagus and a frequent one in mega esophagus.

A third cause of chronic esophagitis is the congestion due to chronic venous stasis resulting from cardiac decompensation, pulmonary sepsis, or particularly portal hypertension from cirrhosis of the liver or thrombosis of the splenic vein.

There may also be secondary or accessory causes such as occupational hazards, notably the ingestion of fine particles of metal or porcelain by metal polishers or porcelain workers, and chemical effects such as that resulting from regurgitation of highly acid gastric juices. In the latter case many times there is undoubtedly a constitutional predisposition (Chapter 15).

In some instances local trauma, particularly the effect of pressure by an indwelling nasogastric tube, may be a contributing cause.

Pathological Anatomy

Chronic esophagitis, like any chronic inflammatory process elsewhere in the body, may appear as an acute or subacute process arising from irritative disturbances of more or less prolonged duration. These may be the action of physical agents or chemical irritants, bacterial invasion as with syphilis or tuberculosis, or involvement by various fungus infections like actinomycosis. As with acute esophagitis, purely inflammatory changes predominate but chronic inflammation, especially when there is venous stasis, becomes complicated by sclerosis which is a frequent sequel. This is characterized by thickening of the epithelium, polypoid or papillary hyperplasia, lymphocytic infiltration and hypertrophy of the submucosal muscular, and glandular layers, venous hyperemia, and interstitial hemorrhage throughout (Fig. 226).

These changes predominate in the areas which are particularly subject to irritation. They may become complicated by the occurrence of acute exacerbations and may lead to ulceration or even areas of necrosis.

These manifestations are characteristic of exfoliative esophagitis as described by Reichmann and by Patterson. Under the microscope it is obvious that several elements of the esophageal wall are involved at once. In the mucous membrane layer the most characteristic change in long standing chronic esophagitis is leucoplakia. It is well known that leucoplakia is represented in dermo-papillary mucous membrane layers by the abnormal presence of a granular infiltration with abundant hyaline changes diffused throughout between the Malpighian bodies and the extremely thickened cornified layer (Fig. 228).

Certain authors have considered this particular condition to be a transitional phase from chronic inflammation to cancer, as though the leucoplakic process



FIGURE 228 Photomicrograph. Leucoplakia of the esophagus ($\times 180$) granules of keratin, hyaline and eleidine at *a*, *b*, and *c*; papillomatosis at *d*, *e*, and *f* (H. L. Guibert.)



FIGURE 229 Photomicrograph in a patient with cirrhosis of the liver and esophageal varices ($\times 30$) At *a*, esophageal epithelium beneath which in the chorion and submucosa four dilated veins engorged with red blood cells may be seen *b*, *c* Glands, *d*, submucosa, *e* muscle bundles (H. L. Guibert)

were a precancerous lesion. Careful study, however, fails to produce evidence that leucoplakic changes are any more frequent in the esophagus involved by carcinoma than in one with no neoplastic changes. In fact, some of the most obvious and far advanced leucoplakic changes are seen in the absence of carcinoma. It appears, therefore, that esophageal leucoplakia is more benign than that which occurs in the buccal cavity unless the importance of the latter may also have been exaggerated. It should be recognized in view of the uncertain knowledge of the etiology of cancer in general that it is difficult to prove any causal relationship between chronic esophagitis and the development of malignant neoplasia.

Another type of change in the mucosal layer is follicular or cystic esophagitis characterized by inflammatory infiltration either of the lymphoid follicles or of the mucous glands of the mucosal layer. This form, which frequently accompanies chronic nonspecific esophagitis, is characterized by the stagnation of secretions in the lumina of the glands and the formation thereby of little cysts. The opening and ulceration of these small inflammatory nodules lead to erosions of the mucosa which look like scratch marks. This follicular type of esophagitis is found characteristically in mega-esophagus.

In patients with esophageal varices, esophagitis leads to a form of chronic periphlebitis of the dilated venous channels (Fig. 229).

Sometimes the arteries and arterioles may show changes in the course of chronic esophagitis characterized by sclerosis and thickening of the vessel wall with a marked tendency to more or less complete obliteration of the lumen. In rare instances there may be also the signs of hyaline degeneration with deposition of calcium typical of the atherosclerosis so often seen in the larger arteries.

Clinical Characteristics

The symptoms of chronic esophagitis may be vague and ill defined. At other times the discomforts characteristic of any esophageal irritation will be felt, namely, a burning sensation during the ingestion of food or sometimes after

eating, retrosternal pain with radiation to the scapular region of the back, a feeling of heaviness in the chest, regurgitation of mucoid secretions, and sometimes hematemesis, usually small in amount

Roentgen Examination

The diagnosis of esophagitis is difficult because the mucosal lesions are often minimal and therefore do not show much change radiologically. Often nothing but an inference can be made and confirmation by esophagoscopy is necessary.

Three degrees of change may be distinguished

(1) In the *first* there is merely the evidence of edema of the mucosal folds. On the roentgen films the folds appear slightly thickened, particularly the major fold which may become as thick as one of the gastric rugae. The distinction, however, is difficult to make.

(2) In the *second* degree of change the mucosal folds appear obviously thickened, irregular, and lumpy (Fig. 230, *A*). The contour of the esophagus is somewhat tubular and slightly irregular along the edges (Fig. 230, *B*).

(3) In the *third* phase of its development the contour of the esophagus is definitely rigid and tubular. Spasm is marked. A slight amount of dilatation may be seen above the diseased segment. Peristalsis is rarely seen in the involved segment because of the lack of normal mobility of the esophageal wall. The mucosa is poorly defined. Its surface is marred by edema and the microscopic erosions with which it may be covered.



FIGURE 230 Esophagograms of patient with chronic esophagitis. Arrows in *A* and *B* point to the irregular appearance caused by the shaggy, roughened mucosal surface.

The roentgenological diagnosis of esophagitis is always difficult to substantiate, and as esophagitis is often associated with malignant stenosis, endoscopic confirmation is always desirable.

Esophagoscopic Examination

Esophagoscopy in the more recent but less severe cases reveals only a certain amount of hyperemia with mucous secretions draped over a mucosa of a reddish or deep bluish-red color. During exacerbations the mucosa is edematous and shows evidence of hypersecretion. Dilated venules may be seen, especially in the upper third. In exfoliative esophagitis the appearance is like that of a second degree burn following the ingestion of a hot liquid. If the swallowing of excessively hot liquids is the cause and if it should be repeated, further destruction of the superficial layers ensues, leading to intermittent exfoliation of the epithelial layer in the form of a thick, ragged pseudomembrane, whitish in color and 0.3 mm. or more in thickness. This phenomenon is often attended by retrosternal pain and a slight amount of bleeding. The exfoliative process may be very slow, however, so that the replacement of the layer takes place beneath the tissue which is to be eliminated. There is usually no evidence of eventual cicatrization and the esophagoscopic appearance after the occurrence is essentially normal.

Leucoplakia is characterized by oval surfaces or clearly delimited raised streaks of a grayish or bluish-white color. The thickness of these areas varies, but the consistency is firm (Fig. 181).

Areas of chronic esophagitis may be sharply localized in one region. In fact, in only about 12 per cent of the cases does it involve the entire esophagus. Of the others, in 75 per cent it is confined to the lower third, and in the remainder it is about equally divided between the middle and the upper third. When it is sharply circumscribed, the striking aspect is the sudden transition from the healthy mucosa to the edematous, thickened, diseased area with perhaps a false membrane and a tendency to bleed easily. Sometimes the two zones appear to be separated by a sharply defined ring.

Differential Diagnosis

A definitive diagnosis between esophagitis and other abnormalities, particularly carcinoma, depends usually upon esophagoscopic examination, often with the performance of biopsy. A careful roentgen examination with special search for evidence of abnormalities of the mucosal folds may serve to distinguish varices, and may reveal the existence of hypertrophic changes in the mucosa of the follicular or papillary type.

Treatment

Unless there is obstructive dysphagia, the treatment should be largely symptomatic. Soothing medicaments such as Gelusil or solutions of bismuth subcarbonate tend to protect the inflamed mucous membrane.

Dietary management includes control of the temperature of ingested liquids to avoid extremes of heat or cold and restriction of intake to liquid or very soft materials, avoiding large particles, spices, seasoning, and roughage.

If the inflammatory process reaches the point where partial obstruction due to swelling and edema begins to interfere with the deglutition of the prescribed diet, it may become necessary to resort to bougienage. This maneuver, however, must be carried out with restraint, stopping with relatively small sizes. Many times gentle dilatation produces a striking degree of relief and one treatment may suffice before the inflammatory swelling subsides of itself. If further treatment should be necessary, the attempt should be made to introduce a slightly larger size bougie than was reached previously. The stretching should never be carried to the extreme of injuring the inflamed area.

It should be emphasized that gentle dilatation of an inflammatory stenosis resulting from edema has nothing in common with the forceful stretching of the kind of cicatricial stricture which results from the healing of erosions and ulceration. In the former instance the effect is more like a form of massage of the tissues and if properly done causes no harm. In the latter no enlargement of the lumen is possible without the tearing and disruption of the scar tissue and, although the immediate effect may be beneficial, the ultimate result is the re-formation of a more pronounced stricture than existed before.

Chronic Esophagitis of the Mouth of the Esophagus (Sideropenic Dysphagia—The Plummer-Vinson Syndrome)

The singular condition best known as the Plummer-Vinson syndrome is so complex that only recently have all of its various aspects become known. It is an affection not strictly limited to the esophagus for, although it is often manifested by an ill-defined and variable form of dysphagia, it affects also the mucous membranes of the lips, the mouth, the pharynx, the vulva, and the anus as well as the skin, the finger nails and toenails, and the constituent parts of the blood.

The condition was first described by Blankenstein in 1893. The characteristic esophageal web or membrane was mentioned by Clark in 1911. Subsequently various additional observations which rounded out our knowledge were made by Bronner, who described the frequency of the disorder in women thirty to fifty years of age who complained of difficulty in swallowing and showed evidences of esophagitis with small ulcerations or fissures, by Mosher in 1917, who emphasized the membrane, by Brown Kelly in 1919, who likewise recognized the characteristic dysphagia occurring chiefly in women and developed the idea of spasm at the mouth of the esophagus as the proximate cause, and by Paterson, who recognized the lesions of the lips and tongue.

The first complete work on the subject was that of Vinson published in 1922 under the title, *Hysterical Dysphagia*. He reported sixty-nine cases, all in women, with two outstanding symptoms, namely, dysphagia and a secondary type of anemia. In twelve of these patients there was enlargement of the spleen. He pointed out that Plummer had already noticed the association of these clinical observations in 1912. Thus arose the paternity of a syndrome which has been given in the medical literature the historically disputable name of the Plummer-Vinson syndrome.

Later the lesions of the tongue, the precocious loss of teeth, and the dryness and atrophy of the pharyngeal and buccal mucosa were emphasized by Moersch.

and Connor in 1926. As the last step in elucidating the complete clinical picture, except for the more recent work of Suzman in 1933, were the contributions of Kuhl and associates on the blood changes and the study of the nailbed lesions by Waldenstrom.

It is interesting to note, without detracting from the value of the report of Vinson which popularized the knowledge of the condition, that this author insisted that the condition was hysterical while evidence was accumulating that it is purely organic and anatomical.

Etiology

The Plummer-Vinson syndrome occurs predominantly in women in the proportion of approximately four to one. Although it has been recognized in a few instances in infancy and childhood, the usual age is 50 years or more. There is a high incidence of the condition in Anglo-Saxon and Scandinavian countries. Although a racial susceptibility has been suspected, a careful search of the literature reveals reports of its occurrence among the Negroes in America, the Chinese, and in South Americans whose varied racial backgrounds may be predominantly either Southern European or native Indian.

It is said that Hurst, the father of the term *achalasia*, had never known of the disease before he visited the United States in 1919, and that it was he who proposed the designation *Plummer-Vinson syndrome* upon his return.

Pathogenesis

It is now agreed that the syndrome is caused chiefly by a disturbance of the assimilation of iron by the organism. The outstanding clinical signs and symptoms are always accompanied by an iron deficiency anemia (hypochromic). The mechanism of this sideropenia is not clear, especially since a regimen of low iron intake alone, though it always results in a severe anemia, does not necessarily give rise to the web and other characteristic changes of the Plummer-Vinson syndrome. In pernicious anemia, on the other hand, the patients often experience minor disturbances of the pharynx or esophagus. Failure to absorb a sufficient quantity of iron, possibly because of atrophy of the gastric mucosa has been mentioned.

Accumulated evidence from many sources suggests that the most important factor in the cause of the sideropenia is avitaminosis, especially a lack of the B complex. From the valuable work of Vanotti it appears that on the one hand there is insufficiency of iron catalysts in the cells, namely vitamins B₁, B₂, and nicotinic acid amide, and on the other hand a lack of iron. This avitaminosis provokes the tissue changes and anemia which become manifest in the organs lined by epithelium. A lack of riboflavin is said to be the cause of the cheilitis.

Others have emphasized the possible role of endocrine disturbances, particularly of the thyroid and the suprarenal glands. It has been shown that adrenalin has an inhibitory effect upon ferrous ions. This in turn may interfere with the secretion of gastric juice.

Abnormalities of the function of the vegetative nervous system have been mentioned as a part of the picture, but these, if they occur, are doubtless dependent upon the nutritional deficiency.

It is obvious that, although many advances have been made, our knowledge of the pathogenesis of the condition rests more upon hypothesis than upon actual facts. The ultimate solution of the problem will doubtless come from physico-chemical research bearing upon the activities of the epithelial cells. At present our knowledge is confined to the observation that the Plummer-Vinson syndrome is a clinical complex, the cause of which though still not clearly understood has to do with avitaminosis, endocrine disturbances, and lack of available iron.

Pathological Anatomy

The characteristic changes are confined principally to the mucous membranes and submucosal tissues of the mouth of the esophagus, the anus, and the vulva. Histological examination shows hyperkeratinization of the epithelial layer with marked augmentation in the number of mitoses, desquamation, and, in advanced cases, fibrotic changes. There may also be a certain amount of atrophy of the muscular layers. The fissures of the lips show the changes typical of leucoplakia. The lingual papillae are often absent, and the epithelium over the tongue is smooth and thin. In general the atrophic changes predominate.

The epithelium of the larynx also tends to be atrophic, with evidences of nuclear degeneration of the cells.

The epithelium of the esophagus likewise is atrophic. The muscular layer is infiltrated with connective tissue and numerous round cells. For this reason the dysphagia, which is not hysterical but based upon definite anatomical lesions, is explained as much by the degeneration of the muscle layer as by the presence of a web. The degenerative changes are not confined to the mouth of the esophagus. In fact they often reach the lower portion of the organ as well.

Clinical Characteristics

Although not constant, the most disturbing symptom is dysphagia. MacMillan found that among 1600 patients who complained of dysphagia, the Plummer-Vinson syndrome was the cause in 13 per cent. Its characteristics vary. The usual complaint is of a sensation of constriction in the pharynx which has been designated variously as *globus hystericus*, primary dysphagia, or esophago-spasm. This symptom is accentuated on attempting to swallow solids, especially if they are dry. Liquids are tolerated much better. This paradoxical dysphagia has been attributed to various causes, notably dryness of the mucous membranes, sphincteric spasm, the presence of a web, or ulcerating esophagitis of the mouth of the esophagus. The intensity and frequency of occurrence of the dysphagia vary from patient to patient. It is more noticeable when the patient is fatigued. The evening meal is likely to cause the greatest trouble. It is subject to exacerbations and remissions. An association with gastric ulcer has been established in some patients.

Sometimes the dysphagia assumes the characteristics of acute pharyngo-esophageal spasm. Often it is slow and progressive. Acute attacks may be interspersed with more or less long intervals of normal deglutition. As other manifestations of the disease become more pronounced, the dysphagia may disappear.

even to the point of being forgotten by the patient. Some reports indicate that dysphagia may be absent in as many as 15 per cent of the patients with the Plummer-Vinson syndrome.

There is usually a long history of fatigability. The objective signs usually described are abnormalities of the skin and its appendages and the epithelial mucous membranes. The patient is unusually thin, with an apathetic expression, a pale, sad appearance, and a sallow complexion. The conjunctivae are pale in color. The skin is wrinkled and brownish with a loss of its normal elasticity. The hair is dry. The fingernails are friable and break easily. A pathognomonic sign is the spoon-shaped curve assumed by the nails. Cracks which fail to respond to treatment develop in the skin of the fingertips. Mucosal changes are noticed on the lips, the tongue, the buccal surfaces, the vulva, and the anus. Patients complain of dryness of the nasal mucosa. Fissures form at the corners of the mouth. The lips are thin and inelastic. Salivation is usually diminished. The mucosa of the palate and uvula is dry and pale in color.

The mucosa of the tongue is shiny and smooth, the papillae are atrophic. There are often grooves and fissures in its surface with superficial erosions and leucoplakic changes. Sometimes the tongue is painful and the hypersecretion of saliva induced by the glossodynia makes the fissures of the mouth worse.

The teeth loosen and fall out. Dental prostheses are poorly tolerated. The mucosa of the pharynx is pale, glistening, and smooth, with the appearance commonly spoken of by the disputable term "atrophic pharyngitis". The lesions of the vulva and anus are identical in appearance with those of the mouth but are often complicated by pruritus.

The spleen is sometimes enlarged. Examination of the blood usually reveals a pronounced hypochromic anemia with a hemoglobin often as low as 4.0 to 5.5 grams per cent and a red cell count of 2.5 to 4 million.

Examination of the gastric secretions often reveals achlorhydria. While the complete clinical picture described above often prevails, there are many cases in which the manifestations are incomplete. Some patients may have the dysphagia without anemia and with pathological changes confined principally to the mucous membranes.

Roentgen Examination

In an early stage the membrane or web which is pathognomonic of the syndrome appears as a fine indentation of the anterior wall of the esophagus behind the cricoid cartilage. This shadow is seen usually at the level of the fifth or sixth cervical vertebra. Exceptionally it may be lower, sometimes as far down as the aortic vertebra. The web may occur singly, or multiple membranes may be seen. These tend to slope downward. If the mouthful of barium is large enough, the membrane presents the appearance of a circular constriction, giving the aspect of a thread tied around a soft tube. If the pharynx is not filled with a sufficiently large amount of barium, however, this indentation may not be seen either at fluoroscopy or on the roentgen film.

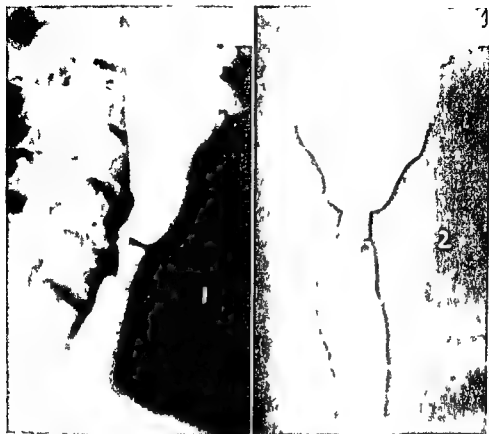


FIGURE 231 Lateral (profile) and anterior views of a patient with Plummer Vinson syndrome showing stenosis produced by a double membrane (After Bonorino Udaondo V D Alotto and H Resano)

In a more advanced stage the membrane projects further into the lumen of the esophagus. It is then easily demonstrated if the pharynx is well filled with barium. The lateral view gives the best visualization although the shadow can be seen, but with more difficulty, in the frontal projection as well (Fig 231).

The examination is always difficult. Radiocinematography may be a valuable aid.

Esophagoscopy

The esophageal speculum should be used instead of the long tube. The web should be looked for on the anterior surface of the esophageal wall. When found it will be noticed that the outer margins of the membrane incline downward on the lateral walls. Sometimes a stenosis in the form of a circumferential diaphragm several millimeters in thickness is encountered. In the absence of an annular constriction the passage of an instrument is easy.

Differential Diagnosis

A diagnosis of Plummer Vinson syndrome should be considered in any woman who complains of dysphagia of long duration. Chronic esophagospasm, chronic esophagitis, and carcinoma must be excluded. The demonstration of the

DISEASES OF THE ESOPHAGUS

pathognomonic membrane by roentgen examination is diagnostic. The esophagoscope is not usually helpful because of the rarity with which the characteristic membrane can be seen. The appearance of carcinoma is usually typical, but unless the growth is of the fungating or polypoid variety it is best to refrain from the performance of a biopsy because of the dangers of perforation.

The characteristic stigmata of the syndrome as it affects the skin and mucous membranes and the hypochromic anemia are helpful in establishing the diagnosis. It should be kept in mind, however, that a carcinoma may develop in the area of involvement at the mouth of the esophagus.

Studies by Darby using a radioactive isotope of iron have made it possible to establish the diagnosis of buccal and lingual lesions due to sideropenia. This form of clinical research will doubtless assume great importance in the study of this disease.

Evolution and Complications

The disease has a tendency to exacerbation and remission, with a tendency to be worse in the spring and the autumn. It is usually of long duration, often twenty years or more.

An important question which must be considered is the possibility that the mucosal changes may predispose to the development of carcinoma. That this may be true is suggested by the well known frequency of occurrence of carcinoma at the mouth of the esophagus in women. Opinions differ as to the coincidence of the two diseases, but it has been reported that 90 per cent of women afflicted with postcricoid carcinoma show a sideropenic anemia. There is strong clinical evidence to suggest that the mucosal changes characteristic of the Plummer-Vinson syndrome are of a precancerous nature. Carcinoma of the tongue, the gums, or the buccal mucosa may be found along with a retrocricoid lesion in patients with the long-standing mucosal changes characteristic of the syndrome.

Treatment

The majority of patients show striking improvement as a result of the administration of iron in large doses. In fact, some clinicians go so far as to say that if the difficulties do not disappear within three months after initiation of the treatment, the diagnosis should be reconsidered. The form in which the iron is given is immaterial. The assimilation of radioactive iron has been followed with the Geiger counter. By this means it has been shown that the liver and the bone marrow are of great importance in the metabolism of iron. It has been proved, also, that the ferrous salts are more rapidly and more completely absorbed than the ferric salts or iron ammonium citrate. The ovate of iron in doses of 0.2 to 0.6 gm. per day has been recommended. Medication should be prolonged to avoid recurrences. Riboflavin or lactoflavin (2 mg. per day) given orally has been administered in recent years with varying results. Vitamin B complex or yeast which is rich in this substance should be tried, but the vitamin therapy is to be considered merely as an adjunct of the administration of iron.

If the anemia is severe, transfusions should be given and the blood hemoglobin level maintained at its proper level by means of iron medication once it has been established.

When a web or diaphragm remains to impede the passage of food through the mouth of the esophagus, mechanical measures must be applied. Dilatation of the orifice with a bougie under direct visual control is usually all that is required. Rarely, it may be necessary to expose the esophagus through a cervical incision for the excision of the web through a longitudinal esophagotomy. (See Chapter 6 on Congenital Anomalies.)

Esophageal Abscess, Phlegmonous Esophagitis, Periesophagitis and Mediastinitis

WHEN THE wall of the esophagus becomes infected with pyogenic organisms, an abundance of which are always present in the contaminated contents of the organ, the inflammatory process may develop either as a localized phlegmon or abscess in the wall of the organ or as a diffuse cellulitis or phlegmonous esophagitis. All degrees of extent and severity of infection are possible, from a relatively harmless localized abscess to an acute fulminating infection ending with extensive mediastinitis.

Etiology

In the vast majority of instances these infections follow some form of injury to the esophageal mucous membrane. The most frequent causes are foreign bodies or some sort of endoscopic or other instrumental trauma. Sometimes, however, bacterial emboli may lodge in the tunics of the esophagus as a result of a bacteremia. These foci may remain superficial and small and may be found only at autopsy as a purulent esophagitis. A similar process sometimes results during the course of ordinary acute esophagitis from infection of the esophageal glands secondary to the occlusion of their excretory ducts. These multiple foci of retained infected secretions may give rise to small abscesses which evacuate spontaneously, leaving innumerable small secondary ulcerations. Finally, in exceptional cases an abscess or phlegmon of the esophagus may occur as a complication of an abscess of the laryngopharynx or the stomach, or a secondarily infected Pott's abscess, of osteomyelitis of the spine, or of acute corrosive esophagitis.

Localized Parietal Abscesses

A typical situation is as follows. After the accidental ingestion of a small foreign body such as a pointed bony fragment or a fish bone, the patient immediately feels a sharp pain which does not abate and is accompanied by progressive dysphagia. In a few hours the painful sensations begin to radiate throughout the cervical region or behind the manubrium, sometimes more on one side than on the other. The pain is accentuated by coughing and by swallowing. In some instances it is of a piercing nature and almost unbearable in intensity. The patient holds the neck immobile and tries to avoid swallowing saliva, coughing, and talking.

Roentgen Examination

One should think immediately under these circumstances of a foreign body, but often such an object because of its nature may not be visible on the roentgen film. *Fluoroscopy*, however, shows an arrest of the thick barium mixture at the site of the trouble.

Esophagoscopy

A zone of edema and reddish discoloration is seen usually in the cervical region a little below the mouth of the esophagus. The foreign body may be found and after its extraction a small ulcerated looking opening is seen from which a cloudy, sanguineous fluid exudes. In other instances a foreign body may not be found. The symptoms persist and disappear only after the spontaneous evacuation through a softening and ulceration of the mucosa of a small collection of frank pus which often has a foul odor.

In cases such as this there is a small submucosal or intraparietal abscess which remains localized around a foreign body or in a minute injury of the mucosa. Usually the lesion does not penetrate beyond the submucosa, remaining superficial and without generalized symptoms. In exceptional cases there are several small foci of pus, each of which empties through a separate orifice giving the appearance of a carbuncle.

Actually these forms ought not to go undiscovered. Since the development of esophagoscopy and the classical descriptions of Von Hacker and others, the diagnosis has become easy especially if the patient gives a history of swallowing a foreign body, particularly if it is a fish bone.

Prognosis

The course is usually favorable. The evacuation of the abscess takes place into the lumen of the esophagus, but in the resulting fissure which is really a small closed cavity it is possible for anaerobes in association with hemolytic streptococci to propagate. In this event, what was originally a small abscess of limited extent may enlarge dangerously toward the mediastinum.

Treatment

Foods and liquids should be withheld and the patient maintained on intravenous infusions. Bed rest should be enjoined and sedatives should be adminis-



FIGURE 232 Drawing made at esophagoscopy showing a submucosal abscess bulging into the lumen of the esophagus

tered to prevent restlessness. An esophagoscopy should be performed but with the greatest care. By this means a foreign body may be found and extracted. If not, search should be made for a bulging collection of pus which might be opened by light pressure of the end of the instrument, the aspirator tip, a long handled knife, or even the end of a sponge holding forceps. A second esophagoscopy may be necessary to accomplish the desired result (Fig. 232).

Antibiotic medication should be employed as indicated until the danger of spreading infection has subsided, at which time the patient can begin oral feedings cautiously.

Phlegmons of the Esophagus

If the foreign body remains buried beneath the mucosa, if treatment was delayed, if unskilled endoscopic manipulations have been carried out resulting in further injury to the esophageal wall, and if the contaminating organisms are exceptionally virulent, the infection may not remain localized. Small pointed bones, especially fish bones, are particularly dangerous because they tend to bury themselves deeper and deeper, thus carrying the bacteria into the outer layers of the esophageal wall.

The cervical esophagus is the most frequently involved because foreign bodies are so often held up there. More rarely the thoracic or supradiaphragmatic segments may be the seat of the trouble. In the cervical region the collection is always located under the posterior wall, probably because the forward arching cervical spine brings the surface into more direct contact with the foreign body. Furthermore, the other walls in this region are more mobile and more elastic.

Pathological Anatomy

Although in circumscribed submucosal abscesses the local signs predominate, in the deeper infections the general symptoms are pre eminent. Several different situations may arise (1) If the collection of pus has not been drained or if it does not evacuate spontaneously, it may increase in volume to the point of causing the walls of the esophagus to bulge, although it does not break through them. This is a true phlegmon of the esophagus. (2) In other instances the collection may escape beyond the layers of the esophagus to invade the periesophageal connective tissues giving rise to a periesophageal phlegmon.

(3) Finally if the infection is not held in check, it infiltrates all the tissues of the esophagus, the periesophageal layer, and the loose connective tissue of the neck and mediastinum, ending up as an acute phlegmonous periesophagitis (Figs 233, 234)

There is, therefore, an indisputable and readily explained analogy between this sort of situation and the pharyngeal and peripharyngeal collections which

FIGURE 233 Drawing of a retro-esophageal abscess in the neck. Typical dissemination of infection in the prevertebral fascial space. (Henke) Abscess shown in dark cross hatching can be seen compressing the esophagus against the trachea

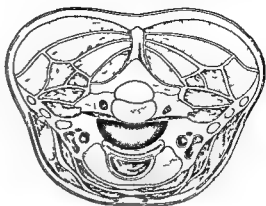
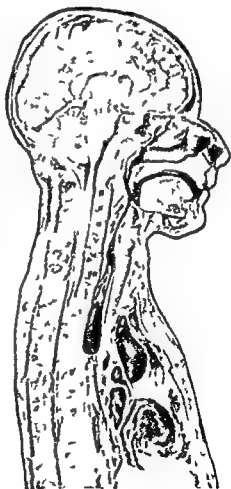


FIGURE 234 Photograph of a sagittal section through the cadaver of a patient who died of mediastinal abscess showing a large accumulation of pus just above the bifurcation of the trachea. The prevertebral space is distended with gas as far as the base of the skull (E. V. Segura)



form in the neck. This subdivision into clinical types corresponds to the cases usually observed in practice, but the clinical manifestations may be rapidly superseded by a fulminating acute mediastinitis which brings about the rapid demise of the patient. In these the infection is invasive and virulent from the beginning.

With the exception of extreme and unusual cases in which, because it is provoked by extensive injury or by unusually virulent organisms, the infection invades the mediastinum with great rapidity, the spread of infection usually progresses only under unfavorable circumstances. An undrained collection of pus or the persistence of a foreign body is the usual condition which causes the infection to penetrate the esophagus and to diffuse throughout the loose connective tissue which envelops the organ. This diffusion usually is most marked behind the esophagus in the space of Henke. If, as is usually the case, the patient is seen late in the course of the disease, and with a large collection of pus already present, it is difficult to establish with precision the exact location of the process whether in the layers of the esophageal wall or in the prevertebral space. The important point is to establish that the infection has not diffused into the loose connective tissues of the neck and mediastinum. In such cases every therapeutic maneuver, whether by a direct approach to the abscess through the esophago-scope or by a transcervical exposure of the organ from the exterior, should be directed toward the discovery and drainage of the pus.

Clinical Characteristics

In the case of a localized phlegmon of the esophagus the clinical picture is usually as follows. There is complete obstruction. Saliva runs from the mouth. The facies is that of a person with severe toxicity. The neck is usually swollen, especially at the base. The patient holds his head immobile and avoids all movement. If the purulent collection is unilateral, the appearance is like that of a patient with torticollis. If it is bilateral, the neck is fixed and straight. The breath is likely to be fetid. There is often a drawing feeling of distress beneath the sternum and attacks of suffocation may occur. The temperature is elevated 39° and 40° C (102° to 104° F). The quality of the voice is altered (faint, nasal or husky).

The cervical region is very painful on palpation. Sometimes there is expectoration of material with an unpleasant odor, necrotic tissue (gangrene) without an elevation of the temperature but with obvious signs of an infection.

The laryngoscopic mirror reveals a marked furring of the tongue, swelling of the posterior wall of the pharynx and edema of the arytenoids.

Esophagoscopy

If the patient's condition permits it, an esophagoscopy should be performed with the greatest care, using a small sized tube. It will be seen that the posterior wall is edematous and if light pressure is made with the end of the tube under direct vision, the collection may be opened, followed by the evacuation of a

large quantity of creamy, malodorous pus. This is a fortunate and rather frequent occurrence.

If the accumulation of pus has not drained of itself or at the time of the extraction of a foreign body, the mucosa over the most swollen area or in a point where necrotic softening appears to have begun is scratched or compressed with a sponge-holder and the pus will escape. In anticipation of this possibility the patient should be placed in the recumbent position before the procedure is begun, ready to be tipped head down when the pus gushes forth.

In cases such as this the escape of a large amount of pus (several ounces or more) indicates that the abscess was actually a collection in the prevertebral fascial space.

Treatment

The principal indication is to evacuate the pus as soon as possible in order to avoid its dissemination downward into the mediastinum because of the effect of gravity or upward toward the base of the skull in those cases in which gas-forming anaerobic infection has taken place.

This is to be done through the lumen if possible. Local anesthesia is employed. In fact, general anesthesia is absolutely contraindicated in the presence of infection because of the danger of spread and particularly of aspiration into the lungs. If the posterior wall is intensely swollen it is best to abstain from the use of the esophagoscope. By palpation with a finger a softened spot may be felt in the apex of the swelling. If pus is obtained from this point by aspiration with a syringe or if a characteristic fetid odor is detected on the needle after it is withdrawn the swelling may be incised at its most prominent point and as low as possible.

If the esophagoscope must be used, its end may be employed to break open the abscess as mentioned above, or the swelling may be incised in its softened apex either with a bistoury or with a curved tonsil knife. The biopsy forceps may also be used.

Sometimes it is better to employ a laryngoscope or a short esophageal speculum in place of the esophagoscope. In every instance the actual opening of the abscess must be done under direct visual control, usually through an area of necrosis at the apex of the swelling.

In the postoperative period the subsidence of the swelling should be closely watched. If dysphagia and pain reappear and if the temperature begins to climb again, a second esophagoscopy must be carried out to drain the collection again.

If an offending foreign body remains the abscess usually drains after the object has been extracted.

If these measures fail, open drainage through the neck becomes necessary. The exposure is made through the type of incision employed for an esophagotomy, using local anesthesia. The retro-esophageal space is entered and the pus is evacuated. Drains should be inserted if need be through both sides of the neck. Sometimes a collar incision may be used and the drains brought out in the supraclavicular space.

Postoperative Care

Oral intake is forbidden, the patient being maintained for several days on intravenous infusions. Recurrences and fistulae must be watched for. Fistulae, however, usually heal spontaneously after a more or less prolonged period of recuperation. Sometimes because of the pressure of the intense swelling of the tissues of the neck it is necessary to perform a tracheostomy.

Appropriate antibiotic medication should be employed from the first. This can be carried out more intelligently and effectively if the sensitivity of the virulent organisms to the various antibiotics can be determined by special cultural techniques such as are available in the laboratories of the larger hospitals. The antibiotic or combination of antibiotics to which the organisms are most sensitive can then be given in large doses until the infection is well under control. It must be emphasized, however, that in spite of the use of antibiotics the infection cannot be expected to subside until the collection of pus is evacuated.

Prognosis

In severe cases with wide diffusion of the sepsis, the prognosis is always grave. Even after the local abscess has been drained and in spite of the use of antibiotics, the patient may succumb to the effects of widespread metastatic infection, often with embolic abscesses in the lungs. This is particularly true with certain resistant strains of *Staphylococcus aureus*.

Diffuse Phlegmonous Esophagitis

In some instances the pus, instead of remaining localized in one area, tends to diffuse throughout the entire length of the esophagus in the layer between the mucosa and the muscularis. This phlegmonous esophagitis may result from the injury produced by a foreign body or from unskilled endoscopic manipulations, from esophageal ulcers, in certain cases of carcinoma, in cicatricial stenosis usually after the creation of a false passage by bougienage, and sometimes without obvious cause.

Pathological Anatomy

The esophagus is thickened throughout its entire length. All of its tissues are infiltrated. The mucosa is lifted from its bed by the pus, and the muscle layers are spread apart. Numerous small islands of pus and areas of gangrene are found. The meshes of the periesophageal connective tissue are distended with a murky fluid. The pleural cavities may contain the same septic exudate.

Clinical Manifestations

The general signs of severe infection predominate. The patient appears prostrated with a tense expression on his face. He may experience chills. He is dyspneic, his pulse rapid and irregular, the respirations shallow. There is profuse sweating. The temperature is usually very high, but in fulminating cases it may be subnormal. Intense pain is felt beneath the sternum, deep within

the chest, in the interscapular region, or at the level of the diaphragm. Dysphagia is complete. Sometimes there is a dry, hacking cough from irritation of the vagus nerves. The patient often dies with a feeling of distress not unlike that seen in cardiac collapse.

In its general aspects the clinical picture resembles the toxemia observed during World War I in gas gangrene infections. Here, also, superimposed acute infection from streptococci and anaerobic organisms serve to complicate the picture. There are, however, various degrees of severity of the infection and not all forms are lethal.

In the most severe cases, which ultimately become complicated by mediastinitis, it may become necessary to perform a mediastinotomy to drain the retro-esophageal tissue space. In this regard it is essential to make a clear distinction between the superior prevertebral space above the level of the tracheal bifurcation and the inferior prevertebral space below that point. In the superior space the connective tissue is very loose whereas from the bifurcation downward this tissue becomes more dense. This tends to limit the spread of infection toward the more dangerous areas in the mediastinum, the pleurae and the pericardium. Collections of pus which accumulate in the superior space tend to spread into the cervical region where they may be exteriorized whereas those in the lower space remain within the mediastinum.

From the anatomical point of view, however, the original conception of Henke that there is physical continuity between the prevertebral cellular tissues of the neck and the posterior mediastinum is correct. The lymph channels according to Rouviere, also intercommunicate. If certain factors are taken into account, such as the effect of gravity, the respiratory movements, and the pulsations of the great vessels it is easy to understand that septic liquids might be drawn from the neck into the mediastinum where the spread of infection in the areolar tissues leads to diffuse mediastinitis.

Mediastinitis, Mediastinal Abscess

Sometimes the reaction in the mediastinal tissues is minimal and the process subsides spontaneously without undergoing suppuration. There is neither pus, septic fluid nor odor even in the presence of a fever. If the mediastinum is opened to provide drainage no localized focus is found. In other instances the infection is of such a fulminating nature that no localization occurs because of the swiftness with which the process reaches its fatal termination. In the latter the tissues are discolored (grayish or greenish) with loss of recognizable structure and have a fetid odor. This situation is often indicative of the presence of anaerobic organisms.

Between these two extremes are the cases in which the infection becomes localized in a mediastinal abscess.

Pathologic Anatomy

In the diffuse phlegmon total invasion of the entire retro-esophageal space is observed. Purulent peribronchial lymphangitis may be recognized by the presence of streaks of a yellowish color extending along the roots of the lungs.

and over the major bronchi. Sometimes the lymphangitis extends into the retroperitoneal spaces and ascites may develop.

In mediastinal abscesses no inflammatory barrier is found. These abscesses rarely extend below the broncho-aortic region unless the source of the invading infection is in the lower esophagus. The aortic arch limits the spread of those which lie above it and tends to cause the process to develop more toward the right pleura or lung than toward the left.

In severe or long-standing cases the trachea may become involved to the point of producing a perforation, suppurative thyroiditis may develop, and rarely septic osteo-arthritis of the cervical spine. One patient observed personally with Guerrier and Causse showed cervical spondylo-arthritis with quadriplegia, there had been perforation of the cervical esophagus by a chicken bone which was extracted twenty-five days after it had been swallowed.

Clinical Characteristics

Three degrees of severity may be observed.

1 The fulminating infections lead to death in a few days with a clinical picture characterized by septicemia, a swinging high temperature, chills, prostration, weakness, and often severe dyspnea from complicating pneumonia. There is usually also marked dysphagia.

2 In the less severe cases there is dysphagia, pain in the neck and chest, fever, leucocytosis, and general evidence of toxicity though not as marked as in the former. These patients usually die but not so swiftly as in the fulminating cases. Sometimes they linger as long as a week or even a month. At other times there is a period of seeming remission when recovery appears to be taking place, followed by a recrudescence and a fatal outcome. Occasionally recovery may follow the evacuation of an abscess into a major bronchus, but this is a rare occurrence.

3 In the relatively benign infections a localized abscess develops which can then be recognized by roentgen examination. These patients often have cough and dyspnea as predominating symptoms. Intense pain may be felt in the chest with exacerbations during inspiration. The pain radiates to the inter-scapular region.

The physical signs are often few. Palpation of the neck may reveal induration or subcutaneous emphysema above the clavicles. The trachea may be displaced to one side or the other. More often it is pushed so far forward that the finger cannot be insinuated between it and the sternum. The movability of the trachea is diminished and attempts to move it are painful. The epigastrium may be rigid and tender to pressure. Percussion over the spinous processes often elicits pain. The clinical picture described is that of an untreated patient or one whose infection does not respond to the administration of antibiotic medication.

Roentgen Examination

Roentgen examination may confirm or reveal the diagnosis but only if a precise technique is observed. In the fulminating cases if the exudation is minimal, as it often is, the examination is negative. Suppurative peribronchial lymph node involvement may be seen as thickening or enlargement of the hilar

shadows. In other cases there may be a widening of the mediastinal shadow with bulging of its contours and an increase in density which obscures the tracheal and aortic shadows. In the lateral view there may be an enlargement of the prevertebral space. Localized mediastinal abscesses give rise to a rounded or ovoid shadow of increased density in either the superior or the inferior mediastinum.

Extension of the process into the lung is suggested at first by the presence of a localized pseudopneumonic shadow in the portion of lung adjacent to the mediastinal shadow. This may remain unchanged until a fistula into a major bronchus develops. If this complication should occur air enters the abscess cavities of both the lung and the mediastinum and with the patient in the erect posture a fluid level may be seen in each.

Extension of the process through the pleura may give rise to a generalized or localized pyopneumothorax depending on whether or not adhesions are present. Ingested Lipiodol usually outlines the tract by which the infection has spread. Many times the infection has spread into the extrapleural tissues but the shadow appears much the same as when the pleural cavity is invaded unless Lipiodol studies are made which will reveal its true location. This measure may save the surgeon the experience of entering an uninfected pleural space during attempts to drain the abscess.

Esophagoscopy

It is doubtful if esophagoscopy should be employed in any patient with a mediastinal abscess. The location of the perforation can be discovered by this means however, and in some instances a foreign body may be removed. It is probably preferable to provide external drainage of the abscess first.

Differential Diagnosis

The diagnosis of a latent or actual infection of the mediastinum should be considered if generalized evidences of infection develop after any form of injury to the esophagus. The occurrence is not infrequent but the diagnosis is often unsuspected unless the possibility is kept in mind. If no antecedent cause can be discovered in the patient's clinical history however the diagnosis may be very difficult. The condition may then be confused with septicemia or with a severe pulmonary infection. The roentgen examination must be relied upon to confirm the diagnosis in any case.

Prognosis

Unless the infection becomes localized and presents through an intercostal space where it may be drained by a minor incision the prognosis is usually serious. It has become increasingly obvious that prompt drainage of the mediastinum is highly desirable as soon as evidence of localization is available.

A small localized collection may be drained endoscopically if the pus does not evacuate into the esophagus spontaneously. The moment the infection has spread widely into the connective tissue spaces of the neck or mediastinum however adequate external drainage must be provided if a good result is to be obtained. Even then when the invading organisms are of a virulent type it is

CHAPTER 15

Nonspecific Ulcerations

Contact Ulcers of the Mouth of the Esophagus

The existence of ulcerations in the zone of transition situated between the end of the laryngopharynx and the beginning of the esophagus was recognized as early as 1850. These ulcerations lie symmetrically opposite each other on the anterior and posterior surfaces of this segment. The original hypothesis proposed to explain this phenomenon is still tenable.

As a result of the more or less constant tension of the cricopharyngeal sphincter, the cricoid cartilage and its fibromuscular investment lie closely applied against the opposite mucosa of the hypopharynx. In the course of certain diseases, after certain types of surgical intervention, or among elderly persons whose general resistance is weakened, the combination of disturbances of local circulation and of the pressure of a cartilage rendered heavier because of calcification may cause the development of small ulcerations of the mucous membrane. Owing to the poor nutrition of the mucosal tissues which are still further injured because of the pressure of the cartilage against the underlying vertebral column, these ulcerations increase steadily in size and form the decubital scars called by Chevalier Jackson contact ulcers.

The pathogenesis is often complex. Superimposed upon the poor blood supply of the tissues, other factors may come into play, such as deformities of the vertebral column or the presence of an indwelling tube. Pressure caused by a nasogastric tube, such as the Levin or Miller-Abbott tubes used in abdominal surgery or after resection of the jaw, is a common inciting cause in the presence of local susceptibility of the tissues themselves as outlined above. The additional presence of a tracheostomy tube may produce ischemia sufficient to bring about the formation of an ulcer of this type.

Histologic studies reveal merely the usual evidences of chronic inflammation and local tissue necrosis.

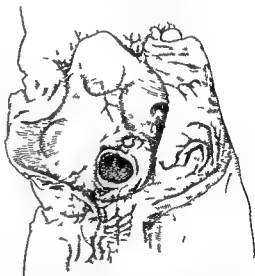
Clinical Characteristics

The presence of a postcricoid ulcer may not be recognized clinically. It is usually manifested by dysphagia with or without respiratory difficulties, but these signs may be obscured by those of the underlying disease. The ulcer is often discovered only at autopsy.

The patient usually complains of intense pain in the throat. He may become hoarse or aphonic. Dysphagia is notable partly because of swelling and rigidity of the tissues at the mouth of the esophagus and partly because of the pain which results from the least effort to swallow. Esophagospasm is a frequent occurrence. In some instances it becomes intense and very painful (pharyngeal tenesmus). A phlegmonous infiltration of the upper esophagus and hypopharynx may develop.

Although the ulcer is not of itself a lethal condition, these often debilitated patients frequently succumb to their basic disease or its complications. At

FIGURE 235 Drawing (at autopsy) of a contact ulcer of the mouth of the esophagus (a common location for so-called decubital ulceration.) (After Wessely.)



autopsy one usually finds an ulcer crater of variable dimensions over the cricoid cartilage which is often denuded by the erosion of the ulcer (Fig. 235). Opposite the first there is usually a second ulcer of identical appearance which often has uncovered the surface of the vertebral body beneath.

Treatment

In debilitated patients treatment is usually ineffectual. If the underlying disease is one from which recovery might be expected to occur possibly with the aid of antibiotic medication or if the patient shows signs of recuperation from a surgical operation, these ulcers may be expected to heal. Prevention however is the best treatment and in every aged or debilitated patient the use of an intubating tube should be confined to the absolute minimum.

Peptic Ulcers of the Esophagus

Sometimes in the thoracic segment but more often in the lower third in what may be called the mixed esophagogastric zone ulcerating lesions of the

perforation of esophageal ulcers in patients afflicted with central nervous system lesions. Others have recorded similar experiences since then.

Finally, in spite of recent experiments by Friedenwald and associates in which peptic ulcers of the esophagus were produced in animals by bathing a small wound of the mucous membrane with 10 per cent hydrochloric acid, no more is actually known at present about the development of an ulcer of the esophagus than of one in the stomach or duodenum. About all we know is that certain people have an ulcer forming tendency.

In the *second* type of case there is an underlying anatomical factor which seems to exert a predisposing influence. These are the patients who have a congenitally short esophagus with a thoracic segment of stomach (brachy-esophagus). In these it is sometimes difficult to determine whether the ulcer has developed in gastric or esophageal mucous membrane. The anatomical pattern characteristic of this condition, with a tubular segment of stomach in the lower mediastinum surmounted by the cardia and above it the short esophagus, favors the development of inflammation (Fig. 118). Gastritis occurs in the stomach portion and esophagitis may develop above it. The esophagitis or gastritis is sometimes complicated by the development of ulcerations which when they heal, cause cicatrization. This does not mean, however, that the scar tissue thus formed is responsible for the herniation of the stomach. It is quite impossible for the contraction of such a scar to pull the cardia as high up in the mediastinum as it is frequently found to be. The displacement in many instances would have to be 10 cm. or more. Actually, the effect of a cicatricial stricture in any tubular organ, whether it be the common bile duct, the ureter, the urethra, the rectum, the duodenum, or the esophagus, is not so much to shorten the organ lengthwise as to narrow its circumference. The direction of contraction is more circumferential than longitudinal. It must be assumed, therefore, that in the majority of instances the altered anatomical pattern is pre-existent and independent of the inflammatory process. The fact that this situation is found in infants and children lends support to this view (Fig. 119). It should be recalled also as further evidence against the assumption that the shortness of the esophagus is the result of contraction of scar tissue, that in patients with extensive deep chemical burns the esophagus is not short and does not become so.

One should therefore make a clear distinction between peptic ulcer of the anatomically normal esophagus, which is analogous to the same condition in the stomach and duodenum, and the ulceration which occurs as a complication of congenital brachy-esophagus.

Pathological Anatomy

In the majority of instances the ulcer develops in the anterior wall of the abdominal segment of the esophagus. It may, however, involve the lower third above the diaphragm, and cases are on record in which true peptic ulcers have been found as high as the bifurcation of the trachea even in an esophagus of normal length. Although usually single, there may be more than one at a time.

The ulcer is usually a small rounded or ovoid loss of substance having a diameter which rarely exceeds 1 cm. (Fig. 236). The edges are clear-cut im-



FIGURE 236 Deep peptic ulcer of the esophagus. Specimen removed at operation. Note the pronounced evidences of chronic esophagitis with thickening of the esophageal wall and leucoplakic changes in the mucosa. The ulcer is located exactly at the cardia. Essentially normal stomach below.

parting a punched-out appearance. The cavity or ulcer crater, depending on its age, may be relatively deep. Those of recent development involve only the mucous membrane and are shallow. The older ulcers have a deep, indurated bed with firm edges. The sides of the ulcer cavity are grayish in color and consist of the wall of the esophagus and, in some instances, fibrotic periesophageal connective tissue. In the latter the ulcer has penetrated all layers of the esophagus and is prevented from a free perforation by the inflammatory barrier built up beneath it. There is also always a surrounding area of esophagitis and periesophagitis which can be recognized on the roentgen film. In extensive ulcers there may be an irregular longitudinal involvement of the esophageal wall, as though several separate lesions had become confluent over a distance of several centimeters. More often the severe ulcerations come to involve the entire circumference of the esophagus, forming what is in fact a girdle-like ulcer of cylindrical shape. It is this type which invariably ends up with the formation of a cicatricial stricture.

Microscopic examination reveals that the ulcer bed is formed by a smooth hyaline fibrous tissue layer interspersed with muscle fibers and infiltrated with leucocytes. In the edges of the ulcer are small arterioles which are so fixed by the surrounding fibrous process that they remain open when cut. It is these vessels which in certain instances cause massive bleeding.

Clinical Characteristics

The manifestations of so-called peptic ulcer of the esophagus vary according to its severity and duration. Many times the ulcer goes unrecognized and, once the acute phase has passed, the condition may become latent to the extent that it is not discovered until the patient reaches a fairly advanced age. The symptoms include, either alone or in combination, pain, dysphagia with or without regurgitation, and hematemesis.

Pain occurs in at least 80 per cent of the patients. As with duodenal ulcer,

however, pain may be absent so that the diagnosis remains unsuspected until some other symptom or the occurrence of a complication such as obstruction or hemorrhage prompts the performance of a roentgen examination. The pain may be constant. It may be felt with the first swallow, or it may not be noticed until a half hour or more after a meal. It varies in intensity from a slight feeling of heaviness in the epigastrium or beneath the lower end of the sternum to a deep boring pain with radiation to the back and scapular region or sometimes to the hypochondria. The pain tends to occur in periodic exacerbations or attacks separated by intervals of relative freedom lasting several weeks or months.

During the periods when the pain is acute, there may be a point of tenderness on pressure over the epigastrium or the xiphoid process or in the left hypochondrium. It is usually relieved by the ingestion of alkalis, suggesting the role of hyperacidity as a cause.

When the ulcer is active there is often a considerable degree of lower esophagospasm which accounts for an increase in the pain and which may be sufficiently pronounced to cause regurgitation of food just eaten. Actual vomiting does not occur, however, unless there is a concomitant lesion of the stomach or of the duodenum. Because the dysphagia is at first caused only by spasm, it disappears with the subsidence of the acute inflammation as the ulcer heals. As time goes on, however, the repeated healing by scar tissue formation begins to produce a stenosis which results in persistent dysphagia varying in degree with the extent of the cicatricial contraction. From then on the clinical picture is dominated by the complicating stricture, even though ulceration may not be apparent. It has been discovered, however, as a result of resecting the stricture in a number of instances that in a relatively large percentage of the cases a chronic ulcer lies hidden within the stenotic area.

Hemorrhage is a variable manifestation. Frequently it is slight and observed only as small clots or streaks of blood in material regurgitated or as a guaiac-positive stool. Severe bleeding may occur, however, and hemorrhage of this degree is one of the important indications for esophagectomy. Often in these patients the blood leaks into the stomach whence it is vomited, after a large amount accumulates. In that event it has the altered appearance of blood which comes from a gastric lesion.

Roentgen Examination

The chief radiological sign of a peptic ulcer of the esophagus is the finding of a *niche*. This is visible only if the ulcer is deep enough to retain an appreciable amount of opaque medium. If the ulcer is confined to the mucosa only, it is easy to understand that unless it is surrounded by an area of thickening from edema it may not be observed. Furthermore it often happens even in a normal esophagus that a small amount of barium may remain adherent at various points without any ulcer being present. If this should occur in a patient with a clinical history suggesting the presence of an ulcer one can merely say, so far as the roentgen examination is concerned, that an ulcer is probably present.

In a more advanced stage the niche is deeper and the diagnosis is more certain. It can usually be visualized best with the patient in the recumbent



FIGURE 237 Esophagogram showing peptic ulcer of the esophagus in a patient with brachyoesophagus. Evidence of obstruction shown by proximal dilatation. Tubular segment of stomach below the high lying cardia is clearly shown. Note the convergence of the rugal folds of esophagus above and stomach below towards the ulcer crater (Connolly.)

posture. In the majority of instances the shadow is small, rounded, and cone-shaped. It is usually so small that it cannot be mistaken for a diverticulum. The average diameter does not exceed the size of a lentil (0.5 cm.), but cases have been reported of benign ulcers as large as 2 cm. in diameter.

There is often segmental spasm producing a convergence of the mucosal folds toward the ulcer niche which resembles the shadow seen in gastric ulcer (Fig. 237). With ulcers of long duration the element of spasm is less important than the inflammatory edema and scar formation. This is shown by the fact that the narrowing in the vicinity of the ulcer is often several centimeters long, that it is often a little eccentric in the long axis of the esophagus though conserving its regularity of contour, and that it fails to react or at least reacts only slightly to the antispasmodics. These characteristics may be observed in acute cases because of the inflammatory edema of the surrounding esophageal wall which gives the appearance of a stenosis though no scar tissue is present. In such a situation, a small niche is very difficult to demonstrate. These inflammatory stenoses, however, are not as irregular as those caused by corrosive esophagitis. When nothing but a cicatricial stenosis remains after the subsidence of the edematous swelling, the barium passes through a little more easily. There is always, however, a slight degree of dilatation attended by disordered peristalsis which gives way to secondary waves. In some instances the peristalsis proximal to the stenosis is exaggerated.

Esophagoscopy

A recent esophageal ulcer appears as a loss of substance usually elongated in shape, surrounded by a zone of marked hyperemia without annular infiltration or proliferations. The edges are slightly ragged and a little elevated above the

surface. The base or crater of the ulcer is granular in appearance and bleeds easily. Sometimes partially cicatrized and healed areas can be recognized as whitish spots separated from the bleeding granular portion. Sometimes the scars of completely healed ulcers may be observed. Further diagnostic characteristics are that these ulcer scars are usually circumscribed, that they involve only one wall of the esophagus, and that they are easily dilated but bleed freely with each dilatation. It is not easy, however, to distinguish them from the scirrhus type of carcinoma sometimes seen in the esophagus.

Differential Diagnosis

During the acute phase of an ulcer of the lower esophagus it may be very difficult to predict on the basis of the symptoms alone whether the lesion is esophageal or gastric in location. The radiation of the pain from an ulcer in the stomach close to the cardia may be identical with that from the esophagus. Roentgen studies, however, establish the difference.

The differentiation between an ulcer and a carcinoma of the esophagus, however, may be much more difficult. The esophagoscope may not help. Evidence of a proliferating excrescence from the margin of an ulcer usually indicates the presence of a carcinoma and a biopsy of such a lesion is often diagnostic. In other instances, however, the biopsy may be misleading. Many times because of the presence of a zone of inflammation around the margin of a malignant lesion the tissue obtained by the biopsy forceps shows merely chronic inflammation. A deeper bite is necessary in order to secure a piece of the growth itself, but for technical reasons even this may fail. As will be described in the chapter dealing with carcinoma, a higher percentage of positive results is often obtained from cytological studies of saline washings from the vicinity of the lesion.

The differentiation between a cicatricial stenosis caused by peptic ulceration and the scar of a chemical burn depends upon the clinical history and the general appearance as seen on the roentgen film. With ulcer scars the deformity is always limited in extent and located in the lower third of the esophagus. In corrosive esophagitis the upper segments are frequently involved and there are often multiple areas separated by segments of relative freedom from involvement. These areas tend to be more irregular in appearance.

Complications and Prognosis

As with ulcers of the stomach or duodenum, the complications of peptic ulcer of the esophagus are chiefly hemorrhage, perforation and obstruction, the latter with its attendant effects due to malnutrition. Bleeding may consist of chronic oozing of small amounts or frequent repetitions of small hemorrhages, resulting in either event in a chronic anemia. Less often the hemorrhage may be massive with evidences of shock. Occasionally it may be cataclysmic in its violence with rapid exsanguination ending in death. This rare occurrence is usually the result of erosion of the adjacent portion of the aorta by a rapidly penetrating acute ulcer.

Recent acute ulcers also may lead to a perforation into the upper abdomen or more often into the mediastinum depending upon their location. Peritonitis,

subphrenic abscess, mediastinitis, mediastinal abscess, pericarditis, pleural effusion, or even empyema, pyopneumothorax, and gangrene of the lung if it is adherent to the esophagus may be the sequelae of such an occurrence. Erosion into the left auricle of the heart or into the left main bronchus has been reported. It is estimated that penetration through the wall of the esophagus, though not often with a free perforation, occurs in about 15 per cent of the cases.

Although many superficial ulcers involving the mucosa only may heal without leaving a scar, chronic partial obstruction from cicatricial stenosis is a frequent complication in cases of unusually long duration. This will be considered in detail in Chapter 19.

In a large percentage of the cases, however, the patient experiences exacerbations and remissions of the disease over long periods of time, often without suffering any of the more drastic experiences produced by the complications already described. In this respect, as in many others, peptic ulcer of the esophagus bears a striking resemblance in its behavior to the same disease as it is observed in the duodenum.

Treatment

MEDICAL TREATMENT should be relied upon in all uncomplicated cases. Alkalis or soothing demulcents like Gelusil are helpful. The diet should consist of milk and soft, nonirritating semisolids. Extremes of heat and cold should be avoided in the food ingested. Highly spiced or acid foods, and alcohol should be proscribed. Smoking should be avoided. Mastication should be thorough and the patient should be enjoined to eat slowly. The meals should be small and taken five or six times daily instead of three. The teeth should be put in order and dentures secured if needed. Oral sepsis, whether from the teeth or the tonsils, should receive appropriate treatment.

In the majority of instances, medical treatment succeeds in bringing about the healing of uncomplicated ulcers. The patient should be persuaded, however, not to return to his former careless dietary habits and to omit the use of alcohol and tobacco permanently.

As with duodenal ulcer, SURGICAL TREATMENT should be reserved for those patients whose pain cannot be controlled by medical means and those who develop complications such as perforation, hemorrhage or obstruction. In a series of sixty-seven patients operated upon for ulcer and ulcer stricture of the lower esophagus, the principal indication for surgery was as follows:

Intractable pain	11
Obstruction (stricture)	45
Hemorrhage	4
Perforation	1
Combination	6
TOTAL	67

Pain becomes an indication for surgery when, after a reasonably prolonged period of trial with a good medical regimen, it becomes obvious that the patient's suffering cannot be controlled. Often the pain is so persistent that it interferes with the patient's ability to work and lead a normal life. Such patients welcome

the offer of surgical intervention and after the procedure has been accomplished are exceedingly grateful for the relief obtained

The decision regarding the advisability of surgical interference in patients with *obstructive dysphagia* is, however, not so easily made. No difficulty will be encountered if the nature of the pathological process in each case is kept in mind. In many instances the dysphagia is caused by the intensity of the associated inflammatory process. There is often so much edema and infiltration of the esophageal wall that the lumen may become completely occluded. In cases of this sort, if the ulcer is relatively recent and if there have been no previous ulcers which might have left scars and cicatricial stenosis as they healed, the dysphagia will disappear as the acute phase of the inflammation subsides.

In the type of obstruction which results from swelling only, great benefit is often obtained by gentle dilatation with bougies. One or two treatments carefully carried out to avoid excessive trauma and tearing of the tissues may be sufficient to restore a reasonably serviceable diameter to the lumen. The medical and dietary measures are then continued until the process subsides. The technical details of bougienage as a method of treatment of inflammatory stenosis are described elsewhere (Chapter 18).

On the other hand, if the case is one characterized by the repeated occurrence of acute exacerbations and remissions with new ulcerations forming more scar tissue as they heal near the site of former ulcers, the end result is the development of a cicatricial stenosis. Often as time goes on the entire circumference of the esophagus in the diseased segment will have lost its mucosal layer and the formation of a fibrous stricture cannot then be avoided. In this situation bougienage is not only dangerous because of the possibility of perforation of the esophagus proximal to the stricture, but it is also useless as a method of treatment. This is because the enlargement of the lumen is obtained only by means of tearing the cicatricial scar, and the end result is merely the formation of a more dense fibrous contracture. The relief of the dysphagia in cases of this sort may last only a few hours or days so that it becomes difficult or impossible for the patient to maintain a satisfactory state of nutrition.

In every patient with dysphagia complicating an ulcer of the esophagus therefore, a clear distinction must be made between the obstruction due to inflammatory edema which responds kindly to treatment by bougienage and that caused by cicatricial stricture which demands surgical excision.

So far as the *technical details* of the surgical procedure are concerned, there is considerable difference of opinion. Once again less confusion results if the nature of the pathological process is taken into account. Obviously the painful penetrating or actively bleeding, eroding ulcer must be resected. With regard to the cases in which dysphagia is the problem again surgical extirpation is the only successful means of overcoming a cicatricial stenosis.

On the other hand, a stenosis caused by inflammatory edema alone may subside, if not on medical measures alone, after the performance of certain operations other than esophagectomy. These may be the temporary employment of a gastrostomy or jejunostomy to put the lower esophagus at rest, or the performance of a distal partial gastrectomy (subtotal) as advocated by Wangenstein. The reported failures of these procedures notably the latter are un-

doubtedly due to their employment in patients with cicatricial stenosis, which is an irreversible condition. It should be borne in mind, on the other hand, that the recovery experienced by patients whose obstruction is caused by the reversible changes due to inflammatory swelling alone might have occurred if the patient had not been operated upon.

In rare instances when the stricture is localized to a small segment and when an extensive operation is not justifiable because of the age or poor condition of the patient, a limited resection may be performed followed by an end to end anastomosis of the esophagus. Somewhat more often the cicatricial stenosis may be of so limited an extent lengthwise of the esophagus that it can be treated by esophageoplasty, using a longitudinal incision and circumferential closure.

As a purely palliative procedure in a poor risk patient, an esophagojejunal anastomosis may be performed leaving the stricture intact. Another situation in which the latter procedure is advisable is where an extensive subtotal gastrectomy has been performed at a previous time. This situation makes it impossible, either for mechanical reasons or because of the inadequacy of the gastric blood supply after interference with the left gastric artery, to perform an esophago-gastric anastomosis.

All other strictures as well as the bleeding penetrating ulcers should be resected. The method usually employed involves the restoration of continuity of the alimentary tract by the performance of an esophagogastric anastomosis. In the majority of instances this operation is pre-eminently satisfactory and the operative mortality is relatively low (7.5 per cent). A certain proportion of these patients with ulcer of the esophagus, however, all of whom seem to have a predisposition to ulcer formation, have been found to develop postoperative esophagitis, sometimes long after the completion of the operation. The reasons for this occurrence are not always obvious.

One has to do with the altered physiology of the stomach after the operation. It should be emphasized once again that as a part of the procedure in every patient, the condition of the duodenum must be determined. At least 35 per cent of the patients have a coexisting duodenal ulcer or an ulcer scar. Many of them have duodenal stenosis. Because of the gastric atony and pyloric hypertonicity which follow the interruption of the vagus nerves, the performance of an esophagectomy in such patients may result in a high degree of gastric stasis. As Dragstedt has shown, this in turn by stimulating the gastrin antrum brings about an increase in the secretion of acid peptic gastric juice induced by the antral hormonal effect. The end result of these circumstances is both an increased tendency for gastric contents to regurgitate into the esophagus because of delayed gastric emptying and an increase in the destructive properties of the gastric juices which are regurgitated. The avoidance of this situation depends upon maintaining the rapid emptying of the stomach through the pylorus. In many instances, therefore, a pyloromyotomy or better still a Heinecke-Mikulicz pyloroplasty must be carried out as a very necessary part of the surgical procedure.

Assuming that the difficulty in these patients arises from the effects of regurgitated hyperacid peptic juices upon the vulnerable esophageal mucosa, another approach to the problem is the performance of an extensive proximal

gastric resection as a part of the procedure. This is effected by excising almost all the lesser curvature, all of the fundus and a large part of the body of the stomach, leaving a tubular remnant on the greater curvature side with which to perform an end to end esophagogastric anastomosis (Fig 238, 2). This maneuver has proved to be very effective in a small series of patients but it is subject to the inconveniences of a drastic reduction in the volume capacity of the stomach. This aspect, however, can be overcome in the majority of instances.

Other measures which have been advocated with varying degrees of success to avoid postresection esophagitis are the interposition of segments of jejunum or colon between the esophagus and the stomach and complete by-passing of the stomach by the employment of a Roux en Y anastomosis as advocated by Allison. There are technical and physiological objections to both of these procedures. In fact, esophagitis has been observed in patients who have actually had the entire stomach removed.

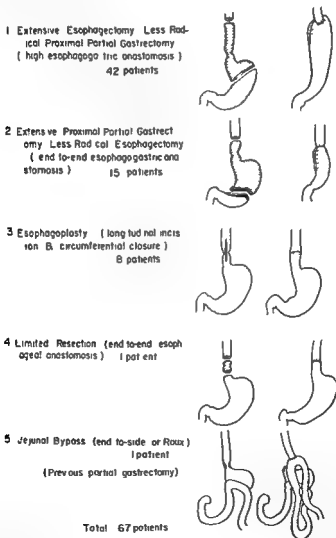


FIGURE 238 Chart showing various surgical procedures employed in the treatment of a series of 67 patients with peptic ulcer of the esophagus in various stages and phases of the disease

One purely technical consideration, though it is described elsewhere (Chapter 20), should be mentioned here. This has to do with the importance of prompt primary healing of the mucous membrane layer. The avoidance of clamps on the resection edges, the use of a sharp instrument to cut the tissues, and the employment of interrupted sutures of fine material (5 0 silk) insure healing in a week or ten days without the development of cicatricial stenosis of the anastomosis. Many times the surgeon blames the disease or some theoretical effect of the physiological alterations produced by the operation without realizing that it is his own imperfect technique which is at fault.

Another approach to the problem of diminishing the occurrence of post operative esophagitis has to do with the assumption that the vulnerability of the mucous membrane of the esophagus may vary from one portion to the other. Several surgeons have observed that the tendency to develop esophagitis is diminished when it has been necessary to remove an unusually long segment of the esophagus with the result that the anastomosis must be made in the upper portion of the thoracic segment. This may be due to the fact that the upper esophagus is nearer the source of the alkaline saliva which might tend to neutralize the regurgitated acid. There may also be differences in the amount of protective mucus secreted by the esophageal glands. This is a factor which doubtless varies from one patient to another. All of these considerations, however, are largely speculative.

As an example of the various methods employed by one surgeon (R H S), the tabulation in Figure 238 is submitted.

RESULTS OF SURGICAL TREATMENT Careful evaluation of the results of surgical treatment (Procedures 1, 2, and 3 in Figure 238) has shown that good to excellent results are obtained in approximately 90 per cent of the patients, with fair to poor results in the remaining 10 per cent.

While it is still premature to make any arbitrary pronouncement, it appears that an extensive proximal gastrectomy with resection of the diseased esophageal segment, cutting across through normal tissues above, followed by an end to end anastomosis, is the most satisfactory method. With it there has been a minimum of complications and sequelae. The importance of performing a pyloroplasty in every instance of associated duodenal or pyloric stenosis cannot be overemphasized.

CHAPTER 16

Specific Infections

Tuberculosis of the Esophagus

Involvement of the esophageal mucosa by the tubercle bacillus is a rare occurrence. For this reason, in spite of some notable work which has been done on the subject, the usual treatise on diseases of the esophagus reserves little room for its consideration. The diagnosis was first confirmed on the basis of autopsy findings in 1837 by Denonvilliers and later by Zenker and Ziemssen in 1877. The first clinical diagnosis with laboratory confirmation and esophagoscopic study was established by Schrotter in 1907. Subsequent reports by various authors in many countries have appeared from time to time until the present.

In autopsy studies made in a general hospital the incidence varies, depending upon the make-up of the series and the thoroughness of the examination, from one in 400 cases to one in 3000 cases. It is more frequently seen in men than in women. It has been observed at all ages from six months to old age.

Pathogenesis

With few exceptions involvement of the esophagus exists principally in patients with far advanced pulmonary disease attended by copious expectoration of sputum full of bacilli. On the other hand in view of the fact that much positive sputum is swallowed by all patients in the active phase of the disease it is strange that esophageal involvement is not more often seen. The usual hypothesis is that the esophageal mucosa is particularly resistant. It is thought for example that the difference between the infrequency of involvement of the pharyngeal and esophageal mucosae and the relatively frequent development of lesions in the intestinal mucous membrane lies in the resistance of the two types of tissue. Other theories have to do with the differences in vascularity, lymphatic supply and mucous secretion. These are all purely hypothetical.

Much discussion has been wasted on the consideration of whether the disease as it affects the esophagus is primary or secondary. The effort to

make this distinction leads only to confusion. True tuberculosis of the esophagus results from the implantation and development of the Koch bacillus in the mucosa of the organ. From this nidus of infection the lesion evolves. No case of infection confined to the esophagus alone, however, is on record.

On the other hand, the tuberculosis of the esophagus which results from the spread of the disease from some local focus in the larynx, trachea, or periesophageal lymph nodes is merely a complication of the laryngopulmonary disease or the lymphadenopathy. In fact, fistulous connections with the respiratory passages may develop and tuberculous nodes may ulcerate into the esophagus without the esophagus itself becoming actively involved. One must make a distinction, therefore, between tuberculosis of the esophagus itself and tuberculous lesions occurring beside the esophagus (para esophageal).

The manner in which the bacillus becomes implanted has been the subject of much study. The possibility of hematogenous spread to the esophagus is largely theoretical. Involvement by way of the esophageal lymphatics is more probable. The lymph drainage from the intramural lymphatic vessels takes place through channels which drain into the peribronchial and paratracheal mediastinal lymph nodes which are so frequently infected. Retrograde spread of bacilli into the esophagus from these nodes may occur. The finding of submucosal tuberculous nodules in the esophageal wall lends confirmatory evidence for this hypothesis.

Implantation by direct contact of organisms contained in the sputum which is swallowed from time to time is probably a more frequent mechanism. A traumatized mucosa and prolonged stasis of material proximal to a stenosis from esophagitis or a cicatricial contracture are important predisposing factors. In the latter instance, malnutrition may play a role by lowering the tissue resistance. The pseudomucinous esophagitis observed in cachectic patients with far advanced tuberculosis doubtless favors the establishment of local foci of infection as a terminal manifestation of the disease.

More easily explained, however, are the cases of tuberculosis of the esophagus which result from spread by local continuity. An example of this is propagation from the larynx to the mucosa of the hypopharynx and mouth of the esophagus. This takes place from ulcerations which may be simple or complicated by perichondritis. Involvement of the esophagus by continuity may result also from the rupture of an adjacent cold abscess of the spine, an adherent, medially placed cavity in the lung, or a mediastinal abscess in infected lymph nodes.

Pathological Anatomy

With the exception of ulcers which spread from the larynx, the most frequent localization of the process is in the midthoracic segment. Three forms may be distinguished: the ulcerating, the hypertrophic, and the granular. The infection, when it involves the esophagus, behaves much as it does in other organs which have a mucosal lining. Ulcerations form and cicatrization results. The only distinguishing characteristic depends upon the fact that the esophagus is a narrow tubular organ.

The ulcerating form is the most common. It may be observed at any level

but there is a preponderance just above the level of the tracheal bifurcation. The ulcers may be single, multiple, or confluent and variable in size. They are usually shallow with ragged edges and a grayish purulent looking bed. The adjacent mucosa is often infected and the ulcer is then surrounded by small tuberculous nodules in various stages of ulceration. Large confluent ulcers tend to be ovoid in shape in the long axis of the esophagus and surrounded by congested infiltrated mucosa (Fig. 239).

Typically, these ulcers are superficial and have no tendency to penetrate beneath the muscular layer. In exceptional cases, however, the ulcer may penetrate deeply into the periesophageal cellular tissues or even into the adjacent trachea to form a fistula. As with the others, the appearance of these ulcers is indolent and their margins are ragged.

From the histologic point of view, the usual evidences of inflammatory ulceration are observed with destruction of the mucosal and submucosal layers and the substitution of a zone of hemorrhagic or purulent detritus (Fig. 240). What remains of the submucosal and other connective tissue and muscular layers of the esophageal wall is more or less completely infiltrated with lymphocytes and in addition numerous polynuclear cells, the result of superimposed secondary infection by pyogenic organisms. Typical tuberculous follicles with the characteristic giant cells are found in the surrounding zone of inflammatory infiltration (Fig. 240). The elastic fibrils are more or less destroyed and the epithelium of the esophageal glands is degenerated. The interstitial tissues show evidence of nonspecific inflammatory infiltration.

Like the former, the *hypertrophic* type of involvement tends to predominate at about the level of the tracheal bifurcation. It may involve only a short length of the esophagus or it may extend 12 or more centimeters. It consists grossly



FIGURE 239 Photograph of tuberculous of the esophagus (ulcerative type) autopsy specimen (After Wessely)



FIGURE 240 Photomicrograph of tuberculous ulcer of the esophagus ($\times 55$) At *a* remains of the epithelium most of which has been destroyed, *b*, ulcer crater, *c* granulation tissue *d e* tuberculous follicles with giant cell at *e*



FIGURE 241 Drawing made from an autopsy specimen stenosing type of tuberculosis of the esophagus

of a concentric, sclerotic thickening of the wall of the organ which causes a marked degree of constriction of the lumen to the point where it is difficult or even impossible to dilate it. The entire thickness of the esophageal wall is involved and often the surrounding mediastinal tissues as well (Fig. 241). Not infrequently the adjacent mediastinal lymph nodes or the trachea are invaded, making it difficult to determine whether the process began in the esophagus or whether it had its origin in the mediastinal tissues. Proximal to the stenosis the esophagus is usually dilated and its mucosal layer is pale in color and dotted with small ulcerations from which pieces of tissue obtained for biopsy may reveal the true diagnosis.

Histologically, this form of tuberculosis of the esophagus often represents actually the end stage of the ulcerating type. It must be conceded, however, that these cicatricial stenosing lesions may not be the result of previous ulceration. In fact, the infection may be confined to the deeper layers of the esophageal wall and characterized by the development of fibrous tissue thickening and cicatrization. The evolution of the lesion in these cases is exactly like that of the hypertrophic form of the disease as it occurs in the intestinal tract.

The microscopic appearance is characterized by the predominance of fibrous tissue proliferation, often with little or no surface ulceration. The appearance is that of an inflammatory granuloma diffusely infiltrated by lymphocytes



FIGURE 242 Photomicrograph of tuberculosis of the sclerosing cicatricial type. At a, b, and c, tuberculous follicles showing one or more giant cells ($\times 200$).



FIGURE 243 Drawing of autopsy specimen showing the miliary form of tuberculosis of the esophagus (After I Villata in *Il Valsalva* 1947, No 7-8)

and histiocytes which have a marked tendency to the production of elastic fibrils and collagen fibers. Although the connective tissue and collagen production predominates, it is always possible to find tuberculous follicles scattered throughout (Fig 242).

The *granular form* of esophageal tuberculosis is the most unusual in occurrence. It is characterized by the presence in the mucosal layer of numerous miliary granulomata of a grayish color but without any tendency to undergo caseation. These often occur in groups in a portion of the mucous membrane, which is heaped up and thickened, the surface covered by verrucous vegetations. Sometimes the surface of such an area is dotted with very small superficial ulcerations (2 to 3 mm in diameter) (Fig 243).

In other cases there is diffuse miliary involvement with many small nodules varying in size from that of a millet seed to that of the head of a pin. These are grayish-white in color and are often arranged in small groups surrounded by congestion of the mucous membrane.

Under the microscope it is seen that the epithelial layer is preserved. In the subepithelial layer there is a diffuse infiltration with lymphocytes and tubercles in different stages of their development. Acid fast stains usually reveal the presence of Koch bacilli in large numbers.

These patients usually present the evidences of widespread or generalized infection including epididymitis, peritonitis, meningitis, pulmonary disease, and renal involvement.

Clinical Characteristics

✓ With the ulcerative type intense pain is the outstanding symptom. This is felt almost constantly but with marked exaggeration during deglutition. Even

the swallowing of saliva is painful. With involvement of the mouth of the esophagus the pain is localized to the throat with radiation to the ears. When the lesion is confined to the midesophagus, it is felt in the substernal region with radiation to the back. It is often felt as a burning sensation. The ingestion of food sometimes brings about temporary relief. The intensity of the pain on swallowing, however, is usually so great that the patient refrains from eating, with resulting loss of weight.

In the hypertrophic cicatricial type of involvement the predominating symptom is obstructive dysphagia. This may appear in a person who seems to be relatively healthy. It undergoes the characteristic evolution of any gradually stenosing lesion, with initial difficulty in swallowing solids ending with the ability to take only liquids. In this type there is usually no pain, but the progression to almost complete inability to swallow may take place in a relatively short period of time.

In the milky form of the disease the symptomatology depends upon the extent and degree of involvement. In severe cases a certain amount of dysphagia may be experienced, but in milder cases there may be no symptoms. Sometimes there may be transitory difficulty with deglutition characterized by inability to eat one meal while the next may be consumed with relative ease.

Roentgen Examination

There is nothing characteristic about the roentgen appearance of tuberculosis of the esophagus. The ulcerative type may display the presence of a niche

FIGURE 244. Esophagogram of a patient with tuberculosis of the esophagus (Dr. Gally).



provided that the ulceration is not too superficial. The hypertrophic form presents the appearance characteristic of any inflammatory stenosis or stricture (Fig. 244). The miliary or granular form shows nothing more than the presence of thickening and diminished peristaltic activity consistent with any inflammatory infiltration of the esophageal wall of comparable degree.

Esophagoscopy

The examination must be carried out with caution and if difficulties such as bleeding are encountered, it is wise to desist from further attempts. Tuberculous ulcers appear superficial with a grayish color and a granular-appearing cavity. When touched with an instrument, such ulcers are unusually painful. The mucosa immediately surrounding appears as a yellowish granular zone, often containing small yellowish nodules representing tubercles. The remainder of the mucosa of the esophagus may appear pale in color.

In the cicatricial type the mucosa appears swollen and edematous. The diameter of the lumen is much constricted. When the stricture is pronounced, the dilated proximal esophagus may be full of pus and the remnants of food.

In the miliary form the mucosa is hyperemic and covered with small granulations the size of cracked wheat particles which are raised above the surface and have a yellowish-white appearance.

Differential Diagnosis

Because of the paucity and diversity of symptoms, the diagnosis of tuberculosis of the esophagus is often difficult. It should be considered in any known tuberculous subject who complains of dysphagia, especially in the presence of laryngeal or pharyngeal involvement. The severity of the substernal pain is a striking characteristic of tuberculous ulcers though of course not actually diagnostic. Esophagoscopic examination, often with the procurement of tissue for a biopsy, must be relied upon. Peptic ulcers, syphilitic ulcers, and the ulcerating form of carcinoma must be excluded in establishing the diagnosis.

With the hypertrophic type, especially in a patient whose pulmonary disease is not active, the problem is to exclude the presence of a carcinoma. The roentgen appearance is sometimes helpful. Many carcinomata present a typical appearance. The scirrhus type, however, is difficult to differentiate from an inflammatory stricture. Reliance must be had on the performance of a biopsy and the cytological examination of esophageal washings. It should be remembered, however, that a biopsy may be inconclusive when the carcinoma lies buried beneath a border of inflammatory tissue.

Cases are on record of the coexistence of carcinoma and tuberculosis, but these are few and far between. An extremely rare case has been reported by Paviot, Levrat, and Guichard in which a tuberculous lesion of the esophagus was complicated by paralysis of the left recurrent laryngeal nerve in the absence of active pulmonary disease.

In the last analysis, as in every other situation, the diagnosis of tuberculosis of the esophagus depends upon the finding of typical tuberculous follicles and, of even more importance, the discovery of the tubercle bacillus in the tissues.

Prognosis

Although instances of healing of the esophageal lesions are on record, the condition is usually terminal and the prognosis is grave. As a rule the patient dies of his pulmonary, peritoneal, or intestinal lesions although the terminal event is of course hastened by the malnutrition provoked by the esophageal involvement. Sometimes death is precipitated by the development of an esophago-tracheal fistula or the erosion of the aorta. Tuberculous meningitis may be the actual cause of death. In general, the downward course of the patient with the ulcerative type of disease is more rapid than that of those who have the hypertrophic type which progresses more slowly.

Treatment

Topical applications of caustic agents like silver nitrate are useless. Antispasmodics may be helpful to overcome esophagospasm. Analgesic agents like cocaine solution may be given orally in case the pain is severe. Antibiotic medication with streptomycin, para-aminosalicylic acid, and isonicotinic acid hydrazide should be employed in full dosage.

In the stenotic hypertrophic form of the disease progressive dilatation may be helpful but the connective tissues are thick and the stenosis is often very tight. If difficulty is encountered, a gastrostomy may be helpful to feed the patient but this should not be done in the moribund.

In general the treatment both hygienic and antibiotic is the same as that for tuberculosis elsewhere and in fact the majority of the patients suffer from advanced pulmonary or generalized disease as well.

Para-Esophageal Tuberculosis

The term *para esophageal tuberculosis* has been applied to the occurrence of fistulae, sinuses and pseudodiverticula on the basis of tuberculous disease in the mediastinal periesophageal lymph nodes. Rouviere has identified a group of lymph nodes in the posterior mediastinum which appears to drain the esophagus. These nodes lie between that organ and the aorta. The nodes lying in the vicinity of the inferior pulmonary veins as well as the subcarinal nodes also drain the esophagus. This group is frequently involved in the childhood type of the disease. As a result of the nodal involvement a marked peradenitis develops which in an early stage merely compresses the esophagus (Fig. 245). In a later phase characterized by the development of an abscess the infection may erode the wall of both the esophagus and the nearby left main bronchus and form a fistula (Fig. 246). There is usually evidence of coexisting pulmonary disease, but the degree of involvement may be minimal.

There is nothing unusual about the microscopic appearance. The tissues show the changes characteristic of fibrinocaseous tuberculosis. The fistulous tract is lined with granulation tissue containing tubercles.

A similar process though less fulminating is responsible for the formation of tuberculous pseudodiverticula of the so-called traction type. The mechanism of the formation of these funnel-shaped deformities has been described previously (Chapter 11).



FIGURE 245 Compression of the esophagus by a mass of tuberculous lymph nodes. Diagnosis proved at operation.



FIGURE 246 Drawing made at autopsy. Tuberculosis of the esophagus showing an ulcer with ragged edges in the anterior wall of the esophagus. Two fistulous openings can be seen at the base of the ulcer crater (After L. G  rry).

Syphilis of the Esophagus

A primary lesion (chancre) in the mucosa of the esophagus has rarely been observed. In the secondary stage of the disease, however, involvement of the esophagus may occur as a part of the generalized infection along with cutaneous and pharyngeal manifestations. Tertiary manifestations are unusual but cause the greatest difficulty.

Syphilis of the esophagus is much more rarely encountered than is tuberculosis. Scarcely more than sixty authenticated case reports are on record in the literature. Furthermore, the intensive and effective antiluetic therapy of the

present makes it likely to disappear completely, especially since it was observed formerly only in the most serious cases and those which were untreated

The disease affects adult males almost exclusively and is almost always of the acquired type. Hereditary syphilis of the esophagus is exceptional. Not over eight or ten such cases have been reported.

Pathological Anatomy

In view of the rarity of the condition, it is not surprising that authenticated cases based upon autopsy studies are even more unusual. It is therefore much to the credit of Guyot of the Institute of Pathology of Geneva that he was able to assemble enough material to write his remarkable study on the pathological anatomy of syphilis of the esophagus for the *Annals of Otolaryngology*. His conclusions are based upon fifty-five observations published in the medical literature between 1917 and 1930 including two additional cases of his own. The latter were the only completely studied cases.

Except for the superficial lesions of the secondary stage which heal in due course the lesions observed, usually at autopsy, are always tertiary manifestations. They are characterized by three principal processes:

- 1 The first is the submucosal gumma or syphiloma which occurs either singly or as multiple foci. These lesions enlarge into the lumen of the esophagus and end in ulceration.

- 2 The second is involvement by the spread from adjacent organs or lymph nodes which lie in immediate contact with the esophagus and as they develop perforate the esophageal wall. If the lesion becomes healed the contraction of the resulting scar tissue may end in stenosis.

- 3 The third manifestation is characterized by scar formation and cicatricial stenosis. This type of reaction is actually in most instances the end stage of the other two. It can, however, occur without the precedence of a gumma. These cases must not be confused with tuberculous strictures and those due to chronic stenosing esophagitis. There is nothing specific about the process as it applies to the esophagus. It resembles the same manifestations of the disease as found in the mucous membranes of the larynx and pharynx.

Macroscopically syphilis of the esophagus can become localized at any level in the organ but the mouth of the esophagus and the normally narrow zones are the points of predilection. When found in the segment just proximal to the cardia the disease has usually spread upward from the stomach.

The lesions are either single or multiple. They are usually accompanied by other luetie manifestations such as epidermal keratosis, gummatous ulcers of the tongue, generalized exanthemata, leucoplakia of the buccal mucosa, interstitial keratitis, periostitis of the humerus, cicatrices of the pharynx, thrombophlebitis of the superior vena cava, syphilitic pneumonitis or more often aneurysm of the aorta.

Microscopically the usual histological changes characteristic of the tertiary phase of the disease are found. The gummatous infiltrations show the diagnostic follicles with giant cells, epithelioid cells and a surrounding crown of lymphocytes. These follicles, however, are rarely typical. The giant cells in particular are often absent whereas they are found more constantly in the tuberculous

and upper esophagus. Sometimes two zones of stenosis may be seen separated by a length of normal tissues.

Differential Diagnosis

The diagnosis of syphilis of the esophagus is difficult to establish. Other signs of the disease such as Argyll-Robertson pupils, absent tendon reflexes, etc., along with a positive serological examination must be found to make one suspicious. Even if all the evidence points to a diagnosis of syphilis, the greatest care must be exerted to make certain that the ulcerating or stenosing lesion in the esophagus is not actually a carcinoma. Otherwise many unfortunate mistakes may be made. Only rarely will a lesion suspected of being a carcinoma, even in a known syphilitic, turn out to be a gumma.

To establish the diagnosis definitely, the serological test must be positive and tissue obtained for biopsy should show the characteristic changes. A positive Wassermann or Hinton test alone, however, means merely that the patient is syphilitic. Furthermore, not too much reliance should be placed on a negative biopsy report because of the difficulty of obtaining tumor tissue beneath the surrounding inflammatory infiltration so often present near a carcinoma.

In addition, because of the generally accepted predisposition of such lesions to favor the development of malignant changes, a known syphilitic ulcer may evolve into a carcinoma.

A therapeutic test of antiluetic medication, by causing a rapid regression of the suspicious lesion, may serve to clarify the diagnosis, but it may do more harm than good when there is a coexisting carcinoma.

Roentgen examination does not provide conclusive evidence for the differentiation.

Other conditions such as peptic ulcer, tuberculosis, and actinomycosis are discussed under the appropriate headings elsewhere.

Complications

Formerly the patient afflicted with syphilis of the esophagus usually died of progressive malnutrition. The most important complication is the development of an esophagotracheal fistula. When this occurs, the unfavorable outcome results not from the syphilis itself but from the pulmonary or mediastinal complications (bronchopneumonia, lung abscess and gangrene of the lung, mediastinitis, etc.).

Treatment

Treatment of the underlying disease must be energetic. In fact, the occurrence of syphilis of the esophagus usually implies that the infection is grave and the case actually neglected. So far as the esophageal lesion itself is concerned, the only treatment required is the bougienage of a cicatrizing stenotic focus using the usual prudent methods, but this is needed only occasionally. If difficulty is encountered, it may be necessary to perform a gastrostomy and to attempt retrograde dilatation. Treatment of a luetic tracheo-esophageal fistula is a difficult matter, but cases are on record in which the fistula has been closed by

surgical intervention, with the interposition of pleura or muscle tissue to prevent recurrence. This must of course be practiced only in conjunction with appropriate antiluetic medication.

Diphtheria of the Esophagus

Diphtheritic involvement of the esophagus mentioned in the classical treatises on the disease has in recent times been the subject of only a small number of communications. The first complete case report was that of Rolleston in 1912. Since then only a few other complete reviews have been made. Furthermore, it appears that the more effective therapeutic measures now available to overcome the Loeffler bacillus are destined to render even less frequent this unusual manifestation of the disease.

Etiology and Pathology

Involvement of the esophagus during the course of a case of diphtheria is very unusual. In fact, after careful evaluation of all the reports on the subject, it is obvious that the condition is rarely seen except in the fulminating form of the disease, particularly in the cases in which the false membrane has a tendency to disperse widely. Even in patients who have died of the disease, esophageal involvement is rarely found at autopsy. The ratio is no more than 1 to 160 cases. Young children are the most susceptible. Only one instance in a patient over fourteen years of age has been reported. It is probably true, however, that in many instances the occurrence goes undiscovered.

The lesion in the esophagus is usually in the upper portion where it appears to be an extension of the involvement of the hypopharynx. There have been cases, however, in which the lower third only was involved, with extension as far as the stomach. Finally, the involvement is sometimes widespread throughout the organ.

The usual *pathological manifestation* is the characteristic false membrane overlying a zone of excoriated mucosa. In about one-fourth of the patients the lesions have an ulcerating, necrotizing character. In exceptional instances the entire cavity of the esophagus is lined with a false membrane which may be thick enough to obstruct the lumen from the pharynx to the cardia.

The Loeffler bacillus cannot always be found, at least according to the older reports.

Clinical Characteristics

The clinical picture devolves into two phases, first the acute infectious period and second the postdiphtheritic stenosis.

ACUTE PHASE In the fulminating cases the symptoms are extremely severe. The patient is in a grave condition and the signs of esophageal stenosis go unperceived. Death supervenes rapidly and the evidence of esophageal involvement appears only at autopsy.

In cases which evolve more slowly the first sign of esophageal involvement is inability to keep down the food taken. In the liquids which are regurgitated one often finds shreds of pseudomembrane and traces of blood. At the same

time the patient complains of severe pain all along the intrathoracic course of the esophagus. When this discomfort is lacking or is not pronounced, it is not always possible to attribute the vomiting to esophageal stenosis. The difficulty is more apt to be attributed to invasion of the pharynx and hypopharynx by false membranes. It must not be forgotten, however, that the patient is always exceptionally ill and usually manifests extensive false membrane formation which goes with an unusually grave form of the disease.

More attenuated and more curable forms of this acute esophageal syndrome in diphtheria may exist, but they are difficult to define. It is possible, however, by careful questioning of patients who develop *postdiphtheritic stenosis* of the esophagus, to elicit symptoms which suggest the extension of the process to the esophageal mucosa during the acute stage. These are cases in which the diagnosis was not suspected during the initial phase of the disease but is established only in retrospect.

The *prognosis* depends upon the general course. Involvement of the esophagus is only one manifestation of the disorder and is usually observed only in the severest cases. Although many patients with such overwhelming involvement die, the occurrence of postdiphtheritic stenosis proves that the prognosis is not universally hopeless.

POSTDIPHTHERITIC STENOSIS The stenosis usually manifests itself rather promptly after the acute phase of the disease has subsided, often as early as three weeks later. The progression, however, is usually gradual. Occasionally a delay of several months to a year occurs before the manifestations of the condition are noticed. This may be explained by the fact that diphtheritic involvement of the esophagus is often localized in the largest segments of the organ. In fact, there seems to be a complete contrast between the cases observed at autopsy in which the lesions are most often localized in the extremities of the esophagus and the cases of patients who live to develop postdiphtheritic stenosis which is often confined to parts of the organ where the diameter is much larger.

Postdiphtheritic stenosis presents no characteristic symptoms. The degree of dysphagia of course depends upon the extent of the cicatricial contraction. Obstruction may become extreme, even to the point where nothing but a ureteral catheter can be passed through. There is usually only one stricture, but this may extend a long distance along the lumen.

Differential Diagnosis

It may be difficult at first to distinguish diphtheritic esophagitis from the effects of paralysis of the esophagus caused by the toxin of the disease (see Chapter 10). In the cases of postdiphtheritic stenoses which do not develop soon after the acute process has subsided, it may not always be suspected that diphtheritic esophagitis is the cause. In such instances all the other usual causes of esophageal obstruction must be thought of and excluded by appropriate diagnostic procedures.

Treatment

The treatment of diphtheritic esophagitis is essentially the treatment of the disease itself. Postdiphtheritic stenosis is treated according to the usual rules

of procedure. Contrary to expectations, however, in consideration of the degree of narrowing often encountered in these patients the bougienage progresses easily and permits a rather rapid restoration of the ability to eat normally. The results are often so favorable that the gastrostomy which may have been necessary at first can ultimately be closed.

Mycotic Infections of the Esophagus

The mycoses usually encountered in the esophagus are actinomycosis, thrush, and sporotrichosis, in that order of frequency.

ACTINOMYCOSIS OF THE ESOPHAGUS

Actinomycosis of the esophagus may develop either as a primary infection of the organ or as a secondary invasion from some adjacent focus. The *primary form* results from actual penetration of the tissues of the esophageal wall by the specific organism of the disease. It may follow the inadvertent swallowing of contaminated fragments of straw or hay, which traumatize the mucosa. In the *secondary form* the infection reaches the esophagus either by propagation from some contiguous focus or by a blood-borne metastasis from some distant site. The disease is caused by *Actinomyces israeli*, which is easily identified by the finding of the so-called 'sulphur granules' in the pus which escapes from fistulae or sinuses in the sputa, or in the material regurgitated. The granules may be seen in sections of tissue.

The 'sulphur granules' are small, irregular, yellowish bodies rarely any larger than 150 microns and consist of a central grouping of close branching filaments of mycelia surrounded by a peripheral halo of rod-shaped hyaline elements. The central core of mycelium can be distinguished from the branching periphery by contrast staining. The organism is anaerobic.

Pathologic Anatomy

The upper parts of the esophagus, namely the mouth of the organ and the broncho-aortic constriction, are the most frequent locations for the disease, but in exceptional instances lesions are found in the lower esophageal segment and at the cardia.

The lesions appear in the form of small primary abscesses of the mucosa which tend to ulcerate deeply and often give rise to fistulae. This is because they usually penetrate all the layers of the esophagus and the organism propagates in the periesophageal connective tissue of the neck or mediastinum with its characteristic formation of sinus tracts, areas of suppuration, and tumor-like swellings.

In advanced cases the infection becomes widespread. Cervical or prevertebral phlegmons may appear. Sinuses may spread through the muscle layers of the back. A widening and thickening of the mediastinum may be seen. Involvement of nearby bony structures may occur. Sometimes the process reaches the pericardium, the heart muscle, the pleura, the lungs, or the brain. All of these things may complicate the original esophageal lesions, the nature of which a

superficial examination might not disclose but which can be discerned as a result of special studies. Sometimes the offending blade of wheat or other substance which initiated the infection is eventually discovered in an abscess or fistula tract.

Clinical Characteristics

The signs of the primary form which are the most important to recognize are scarcely noticeable in the beginning. The early symptoms, namely substernal pain and dysphagia, are indistinguishable from those of any ordinary type of esophagitis. The dysphagia, however, tends to progress much like that due to carcinoma, with very little pain but with excessive salivation and increasing difficulty with the deglutition of solids while the swallowing of liquids goes on unimpaired.

The exact diagnosis is rarely suspected until after the appearance of the involvement of neighboring structures which eventually dominates the clinical picture. The nature of the disease then becomes manifest as the destruction and invasion of the periesophageal tissues, the pulmonary apparatus, and the bony cage of the thorax become obvious. Finally, with the development of fistulae from these areas which discharge the typical sulphur granules, the diagnosis is made.

Esophagoscopy

If an esophagoscopy examination is made when esophageal symptoms are first noticed, a much earlier diagnosis may sometimes be established. By this means the location of the lesions is determined and their nature often correctly diagnosed as a result of biopsy studies which may, however, have to be repeated several times before the true nature of the trouble is defined.

In general, actinomycosis shows lesions of the mucosa in the form of abscesses or ulcerations with ragged edges. There is usually neither actual constriction nor lymph node involvement as there is in carcinoma. Bleeding is unusual.

The pus and the biopsy material show the characteristic granules, which lie in the midst of a network of connective tissue containing polymorphonuclear cells, plasma cells, lymphocytes, histiocytes, and giant cells.

Finally, because of the frequency of relatively early fistula formation involving the air passages, a tracheobronchoscopy and a roentgen examination of the mediastinum are useful measures to locate the lesions and to estimate their extent.

Prognosis

Actinomycosis of the esophagus is a serious disease especially if, as is usually the case, it is not discovered early so that treatment can be started in its incipient stage.

Treatment

Surgical drainage of abscesses is a helpful procedure. If there is a fistula with the trachea or some other portion of the respiratory tract, it may be necessary to feed the patient through a nasogastric tube or by gastrostomy.

The mainstay of treatment however, is medical. Of innumerable substances tried in the past, potassium iodide still holds a place of limited value. The most effective treatment at present is the prolonged administration of sulfadiazine and penicillin in combination.

THRUSH

Any of the diverse forms of *Candida* may in rare instances affect the mucosa of the esophagus. The appearance of this pseudomembranous whitish or greenish-colored coating is brought about by special conditions of debility of the patient and by the reflux of gastric juice which by its acid reaction provokes erythematous lesions on the dry, roughened mucosa. Thrush of the esophagus if unusually severe, can stop the passage of food or even of liquids.

The prognosis and treatment are intimately bound up with the general state of health of the subject, the condition being clinically only an epiphenomenon. Alkaline washes and drinks may be employed with profit.

SPOROTRICHOSIS

Due to the *Sporotrichum schenku* and its related species and rather frequent both in France and America, this affection is characterized by the presence of disseminated nodules in the dermis, the hypodermis, the muscles and under the mucous membranes. Under exceptional circumstances it may involve the chorion of the esophagus exclusively.

The diagnosis is established by culture and by the use of the spore-agglutination test of Widal and Abram.

The treatment consists in the administration of iodides.

Parasitic Diseases

It should be mentioned as a clinical curiosity of unusually rare occurrence that nodules caused by the parasites of *Echinococcus*, *Cysticercus*, *Trichina* or *Filaria* may be disclosed at esophagoscopy.

CHAPTER 17

Disorders Associated with Diseases of the Skin and Mucous Membranes

It is without doubt a triumph of endoscopy to have given us the opportunity to observe the esophageal manifestations of certain dermatological or generalized affections which involve both the skin and the mucous membranes. It is not illogical to assume, for example, that angioneurotic edema which so readily involves the tongue, the pharynx, and the larynx might extend to the esophagus as well. Although such cases are unusual, they are not so much so as is generally believed.

The fact is that the esophageal symptoms which result from this sort of involvement are in general transitory. To be able to visualize the changes which occur, therefore, it is necessary for the endoscopist to be available so that he can make the necessary examination at the very time when the patient notices the symptoms or to have the possibility always in mind and to search for them systematically in every case.

Various observations have demonstrated that esophageal symptoms may be encountered during the course of an attack of urticaria, angioneurotic edema, serum sickness, herpes, bullous pemphigus of the pharynx and buccal mucosa and scleroderma.

Anaphylactic Disturbances

Chevalier Jackson observed a case of *giant urticaria* of the esophagus in a woman who suddenly developed complete esophageal obstruction during an

attack of urticaria of the skin. The roentgen examination showed total obstruction about 6 cm. above the diaphragm. The esophagoscope revealed a whitish tumefaction of the mucosa completely occluding the lumen and obstructing the passage of the instrument. On withdrawing the tube a whitish urticarial streak was observed caused by the rubbing of the instrument against the mucous membrane. Recovery was complete in three days.

An obstruction of the nature described above has been observed in patients with *angioneurotic edema*.

In some instances of *cutaneous urticaria* or of *angioneurotic edema* the mucosa of the esophagus may be found to contain typical-looking urticarial wheals consisting of rounded whitish swellings surrounded by a halo of hyperemia of the mucosa at the base.

Chevalier Jackson has reported an observation of a similar nature in a patient suffering from *serum sickness*, with rapid recovery following the subsidence of the anaphylactic attack.

Herpes of the Esophagus

In this condition the appearance is somewhat different from the above. The esophageal manifestations may or may not be accompanied by cutaneous lesions. The herpetiform vesicles may be seen filled with a yellowish fluid. In a later phase, after the vesicle has broken and discharged its contents the appearance is that of small, irregular, superficial ulcers each with a yellowish bed and surrounded by a bright red halo of inflammation. The similarity of these lesions to those found in the mucous membranes of the mouth is obvious.

Recurrences may be observed after one group of vesicles has healed but in every instance spontaneous recovery is rather rapid.

Pemphigus of the Esophagus

In a few reported cases lesions of the esophagus similar to those which involve the skin and mucosa of the oral cavity have been observed in pemphigus. The esophagus may show bullous swellings of the mucosa with each exacerbation of the disease in the pharynx and buccal mucosa. A patient observed by Tameri had cutaneous pemphigus, complicated by the development of a gradual stenosis which showed on esophagoscopy an ulcerating bulging swelling of the mucosa of the upper esophagus. The process was analogous to that observed in the patient's skin.

Acanthosis Nigricans

Esophageal lesions accompanying this disease have been thoroughly described by C. Pasini (see bibliography).

Scleroderma

In 1916 Schmidt noticed that certain patients afflicted with scleroderma developed dysphagia. Since then observations of esophageal changes in this

disease have been made by various clinicians in many parts of the world. The most important piece of work on the subject is that of Lindsay who in 1949 published his experience of seventeen cases of scleroderma in which lesions of the esophagus were present in fifteen.

It is a common experience for patients being treated by dermatologists for generalized scleroderma to complain rather early in the course of the disease of dysphagia characterized by painless regurgitation, particularly of solids. Liquids usually pass freely.

Pathological Anatomy

Gross examination shows that the mucosa becomes atrophic. The walls of most of the esophagus are smooth with no folds (Fig. 249). Even the major longitudinal fold disappears. In the lower third there is usually a zone of thickening and stenosis. Leucoplakic changes in the remainder of the mucosa may be extensive (Fig. 250). The mucosa and loose submucosal tissues become rigid.

Under the microscope, connective tissue changes identical with those found in the skin are observed (Fig. 251). The muscle bundles and nerves become enveloped by thick connective tissue (Fig. 252).



FIGURE 249 Photograph of a segment of esophagus showing scleroderma. Two perforations are seen (arrows). The upper one is close to the junction of the pharynx with the esophagus. Between the two there is a connecting zone of necrosis with fibrosis of the esophageal wall (J. R. Lindsay).

FIGURE 250 Photograph of scleroderma of the esophagus. The striking plaques of leucoplakia are separated by grooves and superficial ulcerations (J R Lindsay)



FIGURE 251 Section through the esophageal wall in a case of scleroderma showing leucoplakic areas separated by grooves (J R Lindsay)

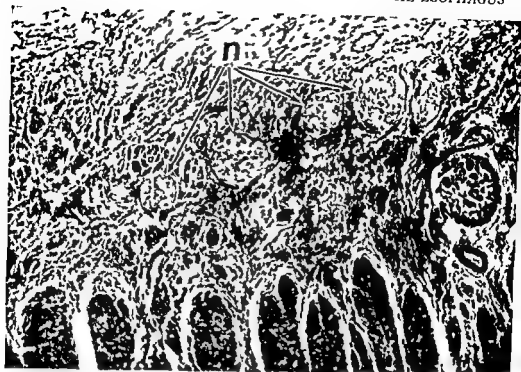


FIGURE 252 Photomicrograph of a section from scleroderma of the esophagus. The muscle bundles and the nerves (n) are surrounded by thick connective tissue (J. R. Lindsay)

It has been maintained by some that these patients tend to have a brachy-esophagus, but if so it is not on a congenital basis. The esophagus becomes short merely because of the retraction of its various layers and of the peri-esophageal connective tissues which become widely infiltrated by dense fibrous tissue having a marked tendency to contraction.

Roentgen Examination

The passage of material through the esophagus is disturbed. The second stage of deglutition begins normally, but a stagnation of five to ten minutes duration sometimes occurs in the lower third of the organ. A film of opaque medium remains attached to the esophageal walls because of the atony of the musculature. There is usually, however, no sign of stenosis except in the cases of cicatricial stricture which have been observed in a few instances.

Peristalsis is markedly diminished. In some instances localized peristaltic activity may be observed in isolated segments where the wall of the esophagus is healthy. Often because of atony of its musculature the esophagus will not empty into the stomach except when the patient assumes the erect position. The organ is asystolic.

Esophagoscopy

Esophagoscopy reveals hypotonicity of the mouth of the esophagus. The mucosa is smooth and whitish in color like the appearance of leucoplakia. Ulcerations may be seen in the affected segment. In fact, the changes are often like those of chronic esophagitis with leucoplakia and ulceration.

Differential Diagnosis

The diagnosis is sometimes confused with carcinoma or the manifestations of chronic esophagitis, particularly the Plummer-Vinson syndrome. The physiological disturbances of the esophagus, particularly of its peristaltic activity, must be differentiated from other forms of esophageal atony or paralysis. Esophagoscopy and particularly the histological identification of the characteristic connective tissue changes in biopsy specimens are necessary to make the diagnosis definite. The condition is always accompanied by cutaneous and other manifestations of the disease.

Death sometimes occurs as a result of perforation of the esophagus or of pulmonary complications due to aspiration of esophageal contents.

Treatment

Treatment is that of the generalized disease without specific reference to the esophagus except with regard to a stricture, but the treatment of strictures resulting from sclerodermal fibrotic changes is no different from that of any cicatricial stenosis. Bougienage may be successful in keeping the passage sufficiently open for the maintenance of adequate nutrition. If this measure should be unsuccessful or if the treatment is abhorrent to the patient, surgical resection can be employed with gratifying results. It is not known whether new strictures will develop following the operation although if the progressive fibrosis cannot be overcome by the newer steroid or other medicinal measures a recurrence at some later date would not be surprising. One such patient has gone six years without experiencing a return of dysphagia.

The above-described esophageal manifestations are still scarcely more than clinical curiosities but it is interesting to recognize that they may occur in order to ascribe the correct cause to certain erstwhile obscure disorders of function of the esophagus which might otherwise be attributed to esophagospasm. From this point of view the personal contributions of Chevalier Jackson are of the highest order.

CHAPTER 18

Acute Corrosive Esophagitis (Chemical Burns)

Etiology

Since 1929 there has been a striking increase in the frequency of chemical burning of the esophagus, particularly in Hungary and in the Balkan countries. This is evident from the number and size of the statistical reports emanating from these areas, particularly those of Belinoff and Metzianu. In France the condition is observed principally in Provence and in Lower Languedoc where a preparation called "olive water" is employed for the operation known as blanching of the olives. Every year after the harvest, during the last months of the year, the hospital at Montpellier shelters numerous patients who have met with difficulty from the ingestion of this caustic material. The same thing is true in Catalonia and in Italy. After 1940 because of the progressive scarcity of detergent materials, the manufacture of substitutes at home brought about a noticeable increase in the number of cases of chemical burning of the esophagus. This was true throughout Europe.

In the United States the accidental ingestion of caustic liquids (usually a solution of lye) occurs most often in rural areas where the domestic manufacture of soap is still practiced to some extent. The incidence, however, is decreasing.

As a method of committing suicide it appears from the statistics of Belinoff that women tend to choose the ingestion of caustic liquids much more readily than men (65 per cent vs. 35 per cent). As an accidental occurrence due to carelessness, particularly in France, chemical burning is seen more often in men and children. The accident is more common in children who are too young to realize the nature of the liquid they are consuming (approximately 58 per cent).

The materials usually responsible for the injury are in order: caustic soda (54 to 70 per cent of cases), sulfuric acid (25 per cent), nitric acid (19 per cent), and hydrochloric acid (2 per cent). More rarely are implicated corrosive sub-

limate, carbolic acid, acetic acid, oxalic acid, iodine solution, silver salts, lysol, gasoline, chloroform, bichromate of potassium and others

Certain authors have incriminated the ingestion of hot liquids, especially in the insane and in children, but in this type of injury the damage is superficial. This is because the heat incites immediate reflex ejection of all but a small amount of the liquid and ingestion of the entire quantity is avoided.

The usual cause of acute corrosive esophagitis is the ingestion of strong caustic solutions.

Except for cases of attempted suicide so common in certain countries (Roumania, Bulgaria, etc.) the occurrence in the vast majority of cases is accidental, especially in children. Efforts have been made to reduce the frequency of these tragedies by regulating the sale of caustic chemicals to householders, but even so it seems to be difficult to control the eternal carelessness of the human race. Special packaging with warnings on the label are helpful but do not eliminate the difficulty completely.

Pathological Anatomy

Present day knowledge of the pathology of acute corrosive esophagitis is based both upon the visualization of the process by esophagoscopy and upon studies at autopsy. Ordinarily the quantity of corrosive material swallowed is not so important as its concentration. Violent pain is usually experienced and immediate vomiting follows. This limits the action of the substance in the lower portions of the esophagus. Solutions of caustic soda and all the strong acids, however, are generally thick and have more the consistency of honey. They descend into the stomach but they tend to remain a rather long time in contact with the esophageal wall and thereby cause deep burns. In the majority of instances the lesions in the esophagus are more severe than those in the stomach. It is only in patients who have attempted suicide and have therefore swallowed a large amount of material quickly that gastric injury predominates. In these the damage done to the stomach may even be the cause of death. When less severe, the injury to the prepyloric area frequently results in pyloric stenosis.

With the exception noted the usual experience is that as soon as the corrosive material reaches the esophagus the lower segment contracts, thus preventing damage to the stomach. It is only when the liquid is unusually thin that it falls directly into the stomach. When this occurs, however, further injury to the esophagus is brought about by the vomiting of the corrosive agent which makes an inevitable second contact with the esophageal mucosa. As the vomited liquid is ejected it often causes burns of the lips, the skin around the mouth, the epiglottis and sometimes the larynx (Fig. 253).

Often the depth of the injury varies from one part of the esophagus to another and sometimes there are areas of complete freedom from injury interspersed with segments where the burn is deep (Fig. 254). These variations depend upon the effects of spasm and peristalsis which hold the material up in one area only to move it on quickly through the next.

In patients who die not long after the accident has occurred the chief finding is a certain alteration of the consistency of the organ, a diminution in



FIGURE 253 Photographs of two patients, each with burns of the lips and chin produced by vomiting of caustic liquid (Belinoff collection)

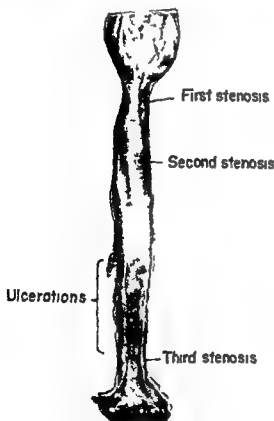


FIGURE 254 Esophagus showing acute corrosive esophagitis (autopsy four weeks after the injury). Stenosis of the esophageal wall can already be seen at the usual locations: the mouth of the esophagus at the level of the tracheal bifurcation and near the cardia. Deep ulceration of the esophageal wall proximal to the lowermost stenosed area.

elasticity, and the occurrence of mucosal fissures. This phenomenon is due to the rapid coagulation of the extracellular fluid which exudes in response to the effect of the caustic. This type of reaction is encountered in all burns of mucous membranes.

The periesophageal cellular tissue is also the seat of inflammatory changes which make it less elastic. This is responsible for the difficulty encountered in the dissection of the esophagus at operation or in its removal at autopsy.

The seriousness of the injury depends not only upon the nature and concentration of the noxious liquid but also upon the quantity ingested. Sometimes

the ingestion of a small amount, even a few drops, when conveyed in the saliva suffices to cause corrosion of the mouth, the walls of the esophagus, and sometimes the stomach, especially when reverse peristalsis brings about the regurgitation or vomiting of the material to cause a second contact with the already injured tissues

The nature and concentration of the corrosive substance also determine the depth of penetration of the injury into the layers of the esophagus as well as its extent along the length of the alimentary tract. Alkalis seem to corrode the esophageal tissues more deeply than acids, which tend to cause more superficial injuries. In the stomach the acids cause more serious damage partly because they are not neutralized as the alkalis tend to be by the acid gastric juices

Macroscopic Changes

All degrees of injury from hyperemia and swelling of the mucosa to complete mortification of the esophageal wall, may be observed

The earliest change is the appearance of *hyperemia* and passive congestion but these are transitory and give way to the development of edema of the esophageal wall which appears soon after the ingestion of the corrosive agent, especially if it is caustic soda. The edema persists with little change throughout the acute phase of the inflammation. It is due at first to the adsorption of the alkali by the tissue cells. This is followed by true inflammatory edema of longer duration

If the liquid is especially strong, the mucosa swells and the superficial necrotic layers become detached from the underlying tissues to form a more or less thickened membrane covering the entire surface of the esophagus. This separates in shreds which are either vomited or passed on into the stomach. The subjacent mucosa is deep red in color with erosions or ulcerations which may vary in depth depending on the concentration and duration of contact of the toxic agent

It is not unusual in severe cases to see the superficial mucosal layer of the entire esophagus cast off and regurgitated like a mold or cast of the interior of the organ (Fig. 255). There is left then only a rough reddish to violaceous colored surface covered with furrows of varying depths and numerous branching small blood vessels

Two kinds of ulcer may be encountered the superficial and the deep. The first involves only the epithelial layer and heals rapidly without treatment. This corresponds to a second degree burn of the skin. Healing is, however, impeded



FIGURE 255 Photograph of a cast of the entire esophagus expelled after acute corrosive esophagitis (Observation of P. Mounier Kuhn.)

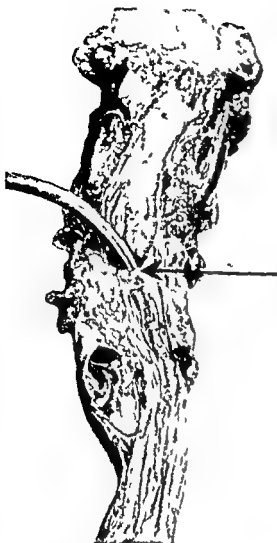


FIGURE 256 Acute corrosive esophagitis caused by ingestion of concentrated nitric acid to commit suicide. Autopsy specimen. Death occurred 52 days after ingestion. Perforation of the esophageal wall followed bougienage (arrow) (Belzoni).

by any mechanical irritation or the application of medicaments. Early bougienage is harmful in these patients.

Deep ulcers involve all layers of the mucosa, the submucosa, and sometimes the muscularis. Occasionally they may extend into the periesophageal tissues. Sometimes a free perforation results.

Microscopic Changes

Studies made in experimental animals which were given various amounts of caustic soda and in patients examined at autopsy make it possible to distinguish two phases of the reaction of the organism against the destructive agent.

✓The first is the period of demarcation which is illustrated in Figures 257 and 258. During this phase, lasting seven to ten days, the necrotic portions become detached, thanks to the activity of the polymorphonuclear leucocytes. These cells form a line of demarcation or barrier between the destroyed tissues and those which remain viable.

The second phase, which actually goes hand in hand with the first, is that of cicatrization. It begins with the appearance of the most intense reaction of

the tissues. This reaction is characterized by the decrease of the polymorphonuclear leucocytes and the development of polymorphous changes in the cells. The line of demarcation becomes more distinct by the development of granulation tissue in which the fibroblasts play a very important part (Fig. 257). At first these cells are round and succulent. Later, from the beginning to the middle of the second week after the injury, they begin to form collagen fibers which are the first elements of scar formation (Fig. 259). The fibroblasts have a dual importance. First they are the most important elements in the filling in of the defect caused by the burn. Secondly, the collagen fibers which emanate from these cells have the property of retracting which diminishes the diameter and eventually obstructs the digestive canal.

The important question is whether or not this role of the fibroblasts can be modified to make it take a desirable form. It is probable that these cells, which possess a considerable degree of elasticity, can be made to do this and that dilatation by bougienage in corrosive esophagitis has no other objective than to modify the activity of the fibroblasts and to give the collagen fibers the

FIGURE 257 Photomicrograph of a section from a patient who died after taking caustic soda with suicidal intent. It shows the histologic appearance of acute corrosive esophagitis. Specimen obtained 8 days after ingestion of the lye. Necrotic tissue already eliminated. Abundant growth of granulation tissue. A Granulation tissue. B vessels in the granulation tissue. C muscle layer. (Belinoff)

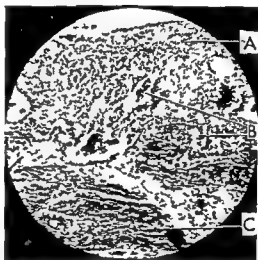
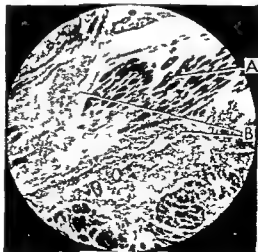


FIGURE 258 Photomicrograph of acute corrosive esophagitis. Patient died 48 hours after ingestion of caustic soda with suicidal intent. Shows the formation of leucocytic barrier. A Necrotic muscle layer. B polymorphonuclear leucocytes which separate the healthy tissues from the necrotic forming a leucocytic barrier. (Belinoff)



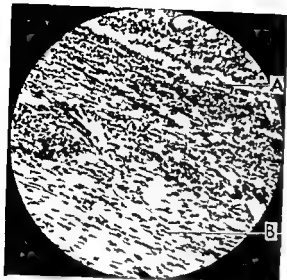


FIGURE 259 Photomicrograph of corrosive esophagitis. Section made 20 days after ingestion with suicidal intent (caustic soda). *A* Granulation tissue. *B* fibroblasts with collagen fibers (Belinoff)

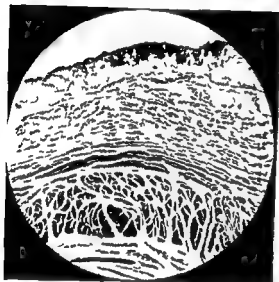


FIGURE 260 Photomicrograph of chronic esophagitis due to ingestion of caustic soda. Specimen obtained 5 years after accidental ingestion. The epithelium rests upon the muscle layer. The submucosal layers, the muscularis mucosae, and the esophageal glands have disappeared. One can picture the lack of elasticity and immobility of such an esophagus (Belinoff)

form and dimensions necessary to maintain the normal contours of the organ being treated

Fibroblasts appear early, actually within twenty-four hours after injury, but the collagen fibers develop much later, usually at the end of the first week and during the second (seven to ten days after the accident). This, therefore, is the precise moment when treatment by dilatation should be started.

The result of the phase of cicatrization is shown in Figure 260. If the burn has not been too deep, the epithelium covers the muscular tunic directly. All the other layers of the esophageal wall, namely the muscularis mucosae, the submucosal tissues, and the esophageal glands, have disappeared. The vestiges of the muscular layer are degenerated and have lost their elasticity and their vitality. As a result of these changes the physiological behavior of such a segment of esophagus is altered. The permeability is decreased, peristaltic movement is abolished, and secretory activity disappears. The prognosis in corrosive esophagitis should always therefore be guarded.

Clinical Characteristics

In severe cases the patient experiences a burning sensation of the lips, the mouth, and the pharynx with exquisite pain beneath the sternum, in the back or in the epigastrium. He appears pale and anxious. Salivation is abundant. The pulse is thready and rapid. If vigorous treatment of the general condition is not started, a state of shock ensues ending in somnolence, coma, syncope, and death.

If death is not immediate, the respirations gradually increase in rate with crises of asphyxia due to edematous swelling of the glottis. The temperature rises. Vomiting of blood or of shreds of mucus may occur. The mouth and lips appear brownish and bloody. The stools are bloody, the urine is scanty and albuminous. Dysphagia is intense. Death results from peritoneal, mediastinal, or pulmonary complications. Often the gastric or intestinal lesions dominate the picture.

In the lighter forms pain is more or less acute in the interscapular region or in the base of the neck or beneath the sternum. With injuries confined to the mouth of the esophagus it has been observed that the pain is felt in the mouth only. The lips, the gums, the internal surfaces of the cheeks, the tongue and the pharynx are hyperemic. The mucosa tends to desquamate.

In cases of moderate involvement after the critical period is passed, the symptoms are the usual esophageal triad of dysphagia, regurgitation, and pain. Every effort to swallow is impossible. Salivation is abundant and the patient often drools at the mouth. The mucous membranes are covered by a membrane which looks like that in diphtheria. The breath is foul. Diarrhea is frequent and the stools are mucoid. The temperature varies. Sometimes there are hiccoughs.

Gradually over the course of the first fortnight the acute manifestations subside and toward the fifteenth day swallowing begins to be possible or even easy. This is the treacherous period. The patient thinks he is cured, whereas in reality the progressive cicatrization of the burned tissues is already under way and eventual stenosis becomes inevitable.

Roentgen Examination

In the acute phase because the changes in the esophagus after ingestion of a caustic liquid are limited to the mucosa and the submucosa the passage of opaque material through the esophagus is slow but not otherwise very abnormal. The mucosal folds, however, are altered and often take on a spiral appearance because of contraction of the longitudinal fibers of the esophageal wall.

More pronounced changes are observed when the stage of stenosis is reached. The mucosal folds are effaced (Fig. 261). During deglutition the liquid stops at the stenosis. The lower limit of the fluid assumes a funnel shape the edges of which are slightly curved inward and the center of which is in the long axis of the passageway. Often there is a long threadlike shadow which indicates the lumen of the esophagus greatly reduced in caliber. Inasmuch as these stenoses develop rather quickly, the degree of proximal dilatation is often very slight. Often at this stage there is a rather marked degree of peristaltic activity.



FIGURE 261 Esophagogram of an early cicatricial stenosis resulting from corrosive esophagitis



FIGURE 262 Esophagogram showing far advanced cicatricial stenosis in a child who swallowed lye. Note marked peristaltic wave in the esophagus above the stricture and evidence of retained food particles

proximal to the damaged segment due to the irritation produced by the injury (Fig 262)

When a gastrostomy has been performed a catheter may be inserted into the lower esophagus for a retrograde injection of barium. This often reveals that the length of the narrowed segment is rather extensive. There are frequently several stenotic regions corresponding usually to the areas of anatomical narrowing at the lower part of the neck, the retro-aortic segment, and just above the diaphragm.

Esophagoscopy

Esophagoscopy performed early in the course of the process reveals in the upper and lower thirds but rarely in the middle third of the organ an edematous mucosa covered by false membranes of variable color usually quadrangular in shape or more rarely circular. The rest of the mucosa is shiny and congested with a dull red color.

In the second stage, ulcerating erosions of variable depth are seen in the mucosa. Later on, after the loss of all the burned tissues has taken place granulation tissue is seen. Later still, areas of scar formation become visible.

Complications

Complications are numerous. They may be immediate or delayed. The most frequent and serious of the early complications is *edema* of the epiglottis, the larynx, and the trachea. The caustic liquid enters the respiratory tract by the aspiration of vomitus, not during deglutition as it was formerly thought. In addition to the local effects upon the mucosa of the air passages secondary infection may develop giving rise to pneumonia, lung abscess, or empyema.

A second complication is *perforation* of the esophagus, which may be spontaneous through an area of necrosis or instrumentally produced (Figs 256 and 263). Periesophagitis and mediastinitis are the results.

Finally, there may be a perforation of the stomach followed by peritonitis, massive hemorrhage from erosion of the aorta (Fig 263), suppurative periesophageal cellulitis, phlegmonous esophagitis, either circumscribed or diffuse and occasionally pericarditis.

The late complications may be the same as those experienced early but occurring during the course of treatment. The principal one is *cicatricial stenosis*, some degree of which develops in 75 per cent of the patients. Thirteen per cent of deaths occur during this period.

The severe cases usually terminate in death. These are usually in suicidal patients who are prone to use very concentrated solutions and to take large amounts. When acids are used for this purpose at least one-half the patients die. After the ingestion of strong alkalis many die within a few hours and about 25 per cent after forty-eight to seventy-two hours. In a personal experience (Terracol), all the suicide attempts (about ten in all) resulted in death within three days at the most. Under these circumstances the medical examiner describes multiple visceral lesions, chiefly perforations of the stomach.

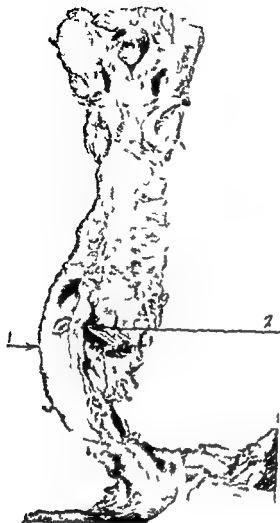


FIGURE 263 Esophagus showing acute corrosive esophagitis with erosion of the wall of the aorta resulting from a perforation. Death from hemorrhage. 1 Aorta 2, perforation of the esophagus

Differential Diagnosis

The clinical history is so characteristic that the diagnosis of acute corrosive esophagitis is easily made. As to the degree and the variety of poisoning as well as the course to follow in each case, the task is much more difficult.

The division proposed by Belinoff into slight cases, those of medium degree, and those which are severe is purely clinical, but it permits an exact clinical diagnosis in each case based upon the findings at esophagoscopy. Early endoscopic examination is safe in the hands of an experienced operator provided that the instrument is introduced without anesthesia and under direct visual control.

This is not true, however, with the blind introduction of an exploratory bougie which is a dangerous procedure because of the risk of perforation, especially since no warning sign exists to make the operator pause.

Prognosis

The prognosis is better in recent years, thanks in part to the employment of the antibiotics. The mortality which was formerly as high as 41 per cent is now

considerably lower. Experience based upon an abundance of case material has brought about certain modifications and corrections in our concepts. Formerly, many of these patients died in a state of shock either soon after or in the first few days following the accident. Those who died later succumbed to the effects of thirst and starvation or as a result of instrumental perforation.

Nowadays the immediate treatment is better carried out than formerly. With the exception of the cases in which the burn is deep, death is unusual, but in these the situation is grave from the beginning because of the necrosis produced by the strong concentration of the caustic liquid. Further improvement in results is due to the fact that treatment is directed according to findings on roentgen examination and esophagoscopy. Between them these methods of investigation reveal the location of the lesions, their depth and their extent and disclose evidence of periesophageal involvement.

Treatment

The physician should learn as much as possible about the circumstances of the accident. It is particularly important to discover the nature of the caustic liquid, a sample of which should be obtained for chemical analysis.

When a large amount of concentrated solution has been swallowed, burns of the stomach predominate and the esophagus assumes a secondary importance. An ice bag may be applied to the neck and another over the hypogastric region. Morphine should be given to control the pain and the usual measures to overcome shock are adopted including oxygen therapy and the transfusion of blood. If the patient can be carried through the immediate danger period the administration of electrolytes and fluid must be continued by the intravenous route. After several days it becomes necessary to provide for the feeding of the patient by gastrostomy or if the stomach is too much damaged by jejunostomy. The end results, however, are not encouraging. The majority of the patients die.

With cases of moderate severity (about 63 per cent, according to Belinoff) the solution may be concentrated but swallowed in smaller amounts. As a rule it is found that the amount ingested is greater than the patient indicates for he believes that it has all been rejected as soon as the error was discovered. With the saliva acting as a vehicle, however, the caustic diffuses rapidly.

After six hours all attempts to neutralize the material by administering dilute acid or alkaline solutions, as the case may be, are useless and the organic secretions have accomplished about all they are capable of by way of defense. If seen before six hours the patient should be made to drink lime water, soap solution, epsom salts or carbonate of soda if the material swallowed was an acid. If an alkali was taken dilute vinegar or citric acid in salt water or lemon juice may be given. For the so-called olive water, olive oil should be administered (Terracol).

Ice may be given by mouth and an ice bag applied to the epigastrium.

If the patient is restless or suffering much pain morphine should be given intramuscularly. If he is in a state of incipient shock with anxiety, faintness and general toxicity, the usual measures are employed as indicated. Dehydration and acidosis are prevented by the use of intravenous injections of appropriate

electrolyte solutions. Fluid may be administered by rectum. The esophagus should be kept at rest. Small doses of dry, powdered subnitrate of bismuth (or subcarbonate) or of kaolin should be administered.

During the next few days the pulse, blood pressure, temperature, and urine output must be watched and the parenteral administration of fluid and electrolytes continued. If the patient can swallow without too much pain, if the pulse and temperature are normal, if there is no dyspnea, hiccough, meteorism, or abdominal tenderness, small amounts of boiled milk, alkaline water, or barium mixture may be prescribed in an effort to soothe the injured surfaces. Topical application of medicaments to lesions of the mouth and pharynx is too irritating and painful.

Complications should be watched for. Hematuria and hiccough are bad prognostic signs.

Even after severe burns, the severity of the inflammatory reaction may be controlled to a large extent by the administration of cortisone. The administration is begun by intramuscular injection as soon as the patient appears for treatment. After the dysphagia subsides, the drug is given orally. Dosage is 25 mg every six to eight hours for children under four years of age. Correspondingly larger amounts are given to older patients. The dose is gradually reduced at the end of the second week and the hormone is discontinued after the third week. ACTH is given during the last few days of the therapy to prevent cortisone withdrawal symptoms.

After the critical period is over, usually by about the tenth or eleventh day, and after the fear of complications is eliminated, every effort must be made to prevent the occurrence of a stricture. A stricture can rarely be avoided without treatment even when only small amounts of caustic have been taken. It is necessary to endeavor to control the cicatricial process in order to prevent any diminution in the caliber of the organ.

There is considerable difference of opinion as to when a bougie should be first introduced. Following the pioneer work of Roux (of Lausanne) published in 1913, certain authorities advocate the *immediate introduction* of a sound which is left in place for two or three weeks. As a modification of this technique, a nasogastric tube (Levin) of rubber or polyethylene plastic may be employed with the advantage that it provides a means of feeding the patient. Others advocate a median course by waiting a few days before introducing a bougie or sound. On the day after the injury, Salzer introduces a hollow elastic bougie filled with lead shot and fitted with a rounded tip. It is softened by hot water to make it pliable. On the second day the instrument is left in place five minutes, on the fourth day thirty minutes. Fever is not considered a contraindication, but the method must be used with caution during the first two weeks.

The results of these modifications of the method are all about the same, namely, 85 to 89 per cent so-called cures.

Belinoff and others do not advocate active treatment until *after a delay* of even to twelve days. Belinoff maintains that before this period bougienage is useless and, especially if carried out blindly, particularly dangerous. This is because the demarcation between viable tissue and that which will be eliminated because of necrosis is not yet established. Furthermore, esophagospasm is likely to be induced and the dysphagia thereby made worse.

Others wait until the temperature returns to normal, at which time a preliminary esophagoscopy is performed, usually on the tenth or eleventh day

Chevalier Jackson has the patient swallow a silk thread soon after the injury for use later as a guide for the treatment by bougienage (see below)

It is important to differentiate between the severe and the less severe cases. In the former, it is best to refrain from any attempt at instrumentation. In the latter, early bougienage is well tolerated or may not actually be necessary. In the cases of medium severity, which are the most important, the decision is difficult. For this the information provided by esophagoscopy is often helpful. In fact, since the invention of the esophagoscope and the development of skill in its use, these injuries can be treated under direct visual control.

The procedure recommended is as follows. Toward the tenth or eleventh day, after the severe initial reaction has subsided, a roentgen examination should be performed to detect areas of spasm or any suspicious looking mediastinal shadows. Then, after cocainization, the esophagoscope is introduced and the wall of the esophagus is examined scrupulously centimeter by centimeter. After this, if no penetrating ulcers are found, an inlying rubber tube of 25 to 28 caliber is introduced. This is usually well tolerated and can be used for feeding. It is left in place for a variable period of from forty eight hours to several days or even weeks. After the expiration of forty eight hours the tube should be tested and, if it is loose and easily moved in the esophagus, a larger one is inserted in its place. This is repeated from time to time until the esophagus will tolerate a size 30 or 32 bougie.

Since antibiotic medication has become available to aid in the prevention of infection superimposed upon the caustic burn of the tissues, it has been possible in all cases to begin the treatment more promptly and with greater safety than formerly. Early treatment is the best prevention of stricture formation. With the elimination of the infection which formerly complicated the picture and added to the extent of the injury, less cicatrization occurs. Penicillin alone or in combination with streptomycin or other antibiotics must be administered in large doses beginning with the first day. The medication is continued until several days after the subsidence of the temperature elevation (usually eight or nine days).

With the subsidence of the fever, if the pulse is normal and the general condition of the patient is good, the dilatation may be begun but not before a study is made of the esophagus under the fluoroscope and of films made during the examination. Moulouguet may be correct when he says that esophagoscopy adds a useless complication in the handling of the treatment and that the view of a swollen bleeding mucosa with here and there plaques of fibrin or brownish eschars does not permit a true evaluation of the seriousness of the injury. On the other hand, if radiographic control is employed, esophagoscopy can be relatively safe. The omission of roentgen examination beforehand, however, is a grave imprudence sure to result even in the most experienced hands in instrumental perforations in a number of patients.

It is impossible to make inflexible rules as to when to start the dilatation. The nature of the injury and the concentration of the liquid must be taken into account. The temperature and general condition of the patient must be favor

able. The roentgen appearance is helpful. The usual time is the tenth to twelfth day, but each patient should be treated individually. By the twelfth day, however, conditions are usually optimal, the critical phase is past, the necrotic debris has been eliminated, and cicatrization has only just begun. With an object which is pliable and easily bent, such as a gum-coated bougie, the esophagitis which gives rise to spasm is eased and the esophagus can be calibrated without danger. The results of immediate calibration are bad, but when suitably delayed early calibration is performed, the results are good.

There are patients, however, in whom all efforts are futile. The temperature remains elevated, the amount of food taken is insufficient, the condition of the burn grows worse. In these, one must not hesitate to perform a gastrostomy without delay. The esophagus, thereby put at rest, soon shows evidence of improvement. The esophagitis subsides, infection clears up, spasm disappears and dilatation with a bougie often along a thread can be commenced relatively early.

The dilatation, once started, should be pursued systematically until a satisfactory caliber has been reached (18, No. 30 to 32). The patient should be warned, however, that it may take a long time to accomplish the desired result and that patience is required. The frequency of the treatments should be regulated with care depending upon the necessities of the case. The liquid or soft solid diet should be prescribed in detail. A regimen rich in calcium and vitamins is necessary, especially in children in whom the general condition, the rate of growth, the weight, and the condition of the respiratory tract must be observed with regularity.

The results of this policy of carefully timed, relatively early but not immediate dilatation in the treatment of chemical burns of the esophagus under the protection of antibiotic medication assures the prevention of stricture formation in a large number of patients. It should always be kept in mind, however, that every esophageal burn demands prolonged close surveillance of the esophageal function, often throughout the life of the patient.

Bougienage in the Treatment of Acute Cases

The location and extent of the injured segment or segments must be established by roentgen examination sometimes supplemented by esophagoscopy. If there is spasm or esophagitis, exploration with a bougie must be delayed until the process subsides. This usually coincides with the improvement of the general condition of the patient. Usually after the routine preliminary preparation, the introduction of the instrument can be attempted under visual control through the esophagoscope. Great care must be exerted to detect the presence of cicatricial bands, blind pouches or recesses, or folds of swollen mucosa, all of which may be discovered by the stagnation of liquids in them. Any of these might be mistaken for the upper orifice of the stenosis which is frequently small in diameter and eccentrically placed. The danger of perforation if this examination is not carried out with perfect accuracy is obvious. Great patience is required of the operator.

This painstaking technique makes it possible to identify and treat many more stenoses in these patients than was possible formerly.

If the swollen stenotic segment is difficult to penetrate, a filiform bougie should be left in place for ten or twelve hours before proceeding further.

If there are several stenoses, one should not attempt to dilate them all at one time. Almost always their several orifices do not lie in a vertical line. The second should not be dilated until the first will permit the passage of the esophagoscope (smallest caliber). If the stenoses are extensive and tortuous, it is inevitable that at the end of several centimeters from the tip of the instrument, the passage of the bougie becomes blind. For this reason it may be wise to carry out the first introduction of the dilator in stages, proceeding part way in the beginning and progressing by degrees at one or more subsequent attempts. In every instance, however, the inflexible rule of *calculated prudence* must be observed.

At the start a daily treatment is necessary and each time the next larger bougie should be inserted, increasing by one number only. The bougie however must penetrate without effort and haste is fatal. Infinite patience is the secret of success in dilating these stenoses. If the stretching is carried too far at any time, esophagitis and its attendant esophagospasm will supervene and the benefits of treatment will be lost at one stroke. It is better always to be satisfied with a small but definite gain each day.

At the beginning of each seance the same size bougie should be introduced as was used the previous time. This should be left in position several minutes before using a larger size. The management of the bougie should not be hasty or disorganized but rather according to a regular, methodical progression. The operator should keep the esophagoscopic appearance of each case in mind. He then has in his fingers, as it were, the memory of the nature of the stenosis, for the initial examination will have established in his mind its position and appearance. It is helpful to compare the location of the aperture of the stenosis to the position of the hour hand of a watch. For example, the opening may be at eleven o'clock, etc. One should know also along which wall the bougie may be made to pass without causing damage, and should be able to recognize each time by the sense of touch imparted from the bougie to the fingers the obstructions, the bands and the openings discovered and localized at the initial esophagoscopic examination. One's memory may be prompted in each case by keeping a diagram of the findings as disclosed by esophagoscopy. Above all a calm persistence, a gentle hand and the avoidance of rough handling in any attempt to overcome an obstruction are absolutely essential.

After a short time it is usually possible to reach a caliber close to normal (20 to 22 mm. at the most in adults and 15 to 18 mm. in children).

Two precautions must be observed (1) The dilatation should never be allowed to become painful, and (2) it should never lead to an elevation in the temperature. If a fever develops the treatment should be stopped and resumed only after the temperature has been normal for several days.

The treatment may have to be repeated at intervals. A reasonable program, after the acute phase is over in about three to four months, is to perform a dilatation routinely at intervals of from every other week to every other month during the first year. Sometimes a monthly dilatation is required.

It must be admitted that there are resistant cases in which the progression

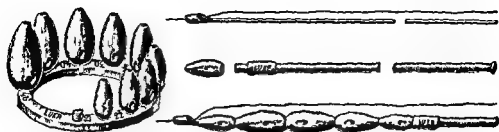


FIGURE 264 Sippy apparatus for dilatation along a thread as a guide Battery of olive tips piano wire metallic thread and flexible introducer

of sizes cannot be pushed beyond No 16 or 17, or in which the gain obtained each time is not maintained. If the patient is one who has been subjected to the performance of a gastrostomy, the retrograde technique may be tried if the peroral method is unsuccessful (See Chapter 4, page 109)

In difficult cases, also, it is useful to employ the method whereby the bougie is guided through the intricacies of a stenosed and damaged area along a string which has been swallowed by the patient previously. This technique was used as early as 1911 by Mixer, Sippy, Plummer, and others. If the patient succeeds in getting the woven silk thread down, the passage of the bougie is usually more easily and more safely accomplished than by any other method, even the direct visualization technique using an esophagoscope. To succeed, however, the string must have passed through the stomach into the intestinal tract by which means it becomes 'anchored'.

The actual technique of this method is as follows. The end of a thread of woven silk (No 2 commercial) about 6 meters in length and thoroughly waxed with beeswax is placed upon the tongue and with the aid of several mouthfuls of water the patient is requested to swallow the thread very slowly at the rate of about 3 cm per hour. At the end of approximately twenty-four hours the thread is usually fixed in position and the dilators can be passed along it. The dilating instrument at first may be a length of piano wire fitted with a tip through which the string may be threaded (Fig 264). After a passageway has been established, one may proceed with the olivary-tipped bougies of graduated sizes which may be attached to a flexible introducer (Fig 264).

This method represents a considerable advance in technique, but it too must be carried out with caution and the need for preliminary esophagoscopy examination remains unchanged.

In patients who have had a gastrostomy, another method is available. This is dilatation on an endless thread which is introduced by means of a filiform bougie, the lower end of which, after its passage through the esophagus into the stomach, is drawn out through the gastrostomy stoma, dragging one end of the thread with it. This end is then tied to the end which protrudes through the mouth. On this loop of thread one may then attach graduated sizes of olive-shaped dilators which may be drawn through the stenosis in either direction, depending upon which gives the better results (i.e., peroral or retrograde).

A second way to introduce the thread which may be preferable to the use of a filiform bougie in some instances, is to attach the end to be swallowed to a lead shot or small sinker. The progress of this object can be observed from

time to time by fluoroscopy and when it has reached the stomach it may be drawn out through the gastrostomy stoma, also with the aid of the fluoroscope. The two ends of the thread are then tied together as above (see also Fig. 273, page 391). In this as in all methods, patience is one of the principal requisites for success.

Results of Treatment

In the favorable cases in which only the epithelial layer is destroyed, complete regeneration of the mucous membrane can be expected and permanent cure is the usual outcome. If all layers of the mucosa, on the other hand, are destroyed, one of two processes may result. Occasionally, where the destruction is confined to small portions of the surface, a sufficient amount of epithelial regeneration may take place to make it possible to maintain an adequate lumen with the aid of gentle dilatation early in the recuperative period. Many of these patients may reach the stage where treatment becomes no longer necessary and they likewise are cured.

It is inevitable, however, that in a certain percentage of patients treatment by dilatation becomes unsatisfactory. This may be because the patient is unsuited by temperament to endure the annoyance of repeated sessions or, more often, it may be because of the severity of the injury. As will be mentioned in Chapter 19, it is only necessary to reflect upon the characteristics of the pathological process to be able to understand the reasons for the failure of bougienage.

When the amount of mucosa destroyed is large and particularly when the entire circumference of the esophageal wall is involved, the type of healing which inevitably takes place always results in the formation of a cicatricial stricture. The granulation tissue which fills in the defect remains uncovered by epithelium and the collagen fibers of its connective tissue component cause an increasing degree of stenosis. Attempts to overcome the obstruction by dilatation are in this instance doomed to ultimate failure because, after a transient and often inconsequential increase in the diameter of the lumen has been accomplished by actually tearing the scar tissue, the end result is the reformation of more cicatrix and the situation becomes worse than it was before. Successful treatment in a case of this sort depends upon surgical resection of the diseased segment of esophagus or the substitution of a segment of intestine or the stomach as a by-pass around the stricture (Chapters 19 and 20).

CHAPTER 19

Cicatricial Stenosis (Stricture)

THE term *stricture* is applied to any unyielding cicatricial stenosis caused by the contraction of scar tissue, in contradistinction to the stenosis which results from edematous swelling, a new growth, external compression, or a congenital anomaly

Etiology

Because of the relatively frequent occurrence of corrosive esophagitis, cicatricial stenosis is the second most common lesion of the esophagus. It stands next to carcinoma in importance. Various causes may be enumerated according to whether they are of inflammatory or traumatic origin.

A stricture may develop as a rare complication of the esophagitis occurring in certain acute infections which are observed nowadays with decreasing frequency. In typhoid fever it may be observed during the convalescent period as a result of an ulceration which may have been unsuspected clinically. Such a stricture is found usually in the retrocricoid area but may be seen in the cervical segment, the narrowing opposite the bronchus and aorta, and in the lower third. The symptoms develop usually at about the fourth week from the onset of the disease but may be experienced by the patient at any time from the second to the twelfth week.

In diphtheria, smallpox, and scarlet fever a cicatricial stenosis is a rare complication, but cases have been observed mostly at autopsy. Young children are the most likely victims.

As already described, strictures may result in certain cases of syphilis or tuberculosis of the esophagus. In actinomycosis the contraction of the peri-esophageal inflammatory tissue may cause a secondary constriction of the esophagus.

Inflammatory strictures may result also from nonspecific esophagitis, peptic ulceration scleroderma, and other related dermatoses, or the late effects of roentgen therapy for thyrotoxicosis or for mediastinal lymph node involvement by tumor metastases. Each of these conditions is described in its appropriate place.

Strictures of traumatic origin include those due to accidental injury (bullet wounds, etc.) or from injury occurring during endoscopic instrumentation. Foreign bodies which may be lodged a long time in the esophagus often provoke an inflammatory reaction ending in ulcerative esophagitis which in turn causes a stenosing cicatrix. As would be expected, these usually occur at the anatomically narrow segments frequently at the aortic constriction. The objects which cause the greatest trouble are those which are exceptionally large or metallic or irregular in contour like fragments of bone, dental prostheses, pins, and so forth. These by causing serious injury to the mucosa may lead to the development of a stricture. The prolonged and often repeated efforts to extract an impacted object of this sort by adding to the local trauma contribute greatly to the unfortunate result.

By far the greatest source of cicatricial stenoses from the statistical point of view is the ingestion of corrosive liquids (see Chapter 18). The frequency of occurrence varies depending upon the availability of these materials. For example, although in France carcinoma is nine times more common than stricture from corrosive esophagitis, in eastern Europe caustic burns are six times more frequently observed than carcinoma. During the German occupation the proportion of lye strictures increased because of the scarcity of soap which prompted many people to make their own. Caustic soda then appeared in almost every household with the result that it was within the reach of everybody including children. In southern Italy and in the south of France, as described in Chapter 18, the liquid known as olive water is responsible for a greater frequency of these lesions in those areas than in others.

Belinoff presents the following statistics regarding the sex and age of 151 patients with stricture from chronic corrosive esophagitis:

<i>Sex</i>	
Male	62 (41 %)
Female	89 (59 %)

<i>Age</i>	
Children 1 to 3 years	41
Children 4 to 7 years	23
Children 8 to 15 years	20
Adults	67

<i>Corrosive Agent</i>	
Caustic soda	82
Sulphuric acid	33
Nitric Acid	29
Hydrochloric acid	3
Unknown	4

In a recent article Franzas reports the agent responsible in 205 cases as

Lye (Caustic soda)	169
Ammonia	11
Acids	24
Potassium carbonate	1

In a personal experience (Terracol) of over 300 patients observed during a twenty-year period, alkaline caustics were ingested in 90 per cent of the cases

A final cause of stricture of the esophagus may be the application of radium or the use of roentgen therapy

Pathological Anatomy

The details of the injury to the esophagus which leads to a cicatricial stenosis have been presented in Chapter 18. In the state of cicatrization the connective tissue element in the granulation tissues becomes more and more dense, undergoes fibrosis, and gives rise to a very tight tough constriction of an annular or tubular configuration. This is the end stage of the chronic corrosive esophagitis which precedes it.

In the stenosed zone, even after several months have elapsed, there are always signs of chronic inflammation of the mucosa and submucosa and usually of the circular muscle layer. The longitudinal muscle layer is less often involved. In the depth of the muscles the nerve fibers and ganglion cells are surrounded by inflammation and finally in the areas above and below the stricture the muscular layer is hypertrophied due to spasm. In summary, the microscope confirms the presence of a permanent basis of chronic esophagitis on which are superimposed exacerbations of acute esophagitis.

The strictures vary from person to person as to their extent and their depth. If the burn reached only the submucosal connective tissue the sclerosis remains limited to the superficial layers. This gives rise to superficial adhesions, bands, swellings, annular folds, and valvular flaps which are merely mucosal or membranous contractures. Often, however, because of superimposed myositis the subjacent muscular layer participates in the process and limits the elasticity and distensibility of the esophagus.

If the muscle was destroyed by the caustic liquid, all the layers of the organ end up by forming a single, hard, impervious, thick layer of fibrous tissue. Sometimes the stenosis is limited in extent like a ferrule, more often it is an extensive tubular obstruction. There may be multiple strictures with segments of uninjured esophagus between them. There is usually a certain amount of periesophageal fibrosis as a part of the process (Figs 265 and 266). The lumen is usually preserved, but not always. In fact, complete obliteration is a more frequent occurrence than was formerly thought.

More and more especially with modern treatment, cicatrization and epithelization of the injured portion take place without serious sequelae but an edematous, infected, ulcerated zone may persist for a long time. This tends to perpetuate the element of spasm and makes endoscopic maneuvers and efforts to dilate the stricture dangerous.

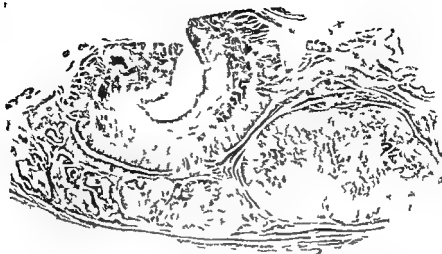


FIGURE 265 Photomicrograph of a section taken from a patient with chronic corrosive esophagitis with stenosis caused by ingestion of lye showing marked periesophagitis (After L. Cojazzi and G. Guran)

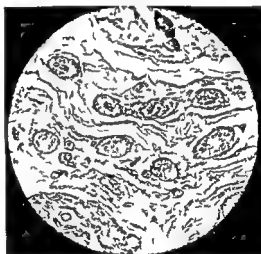


FIGURE 266 High power view of the same case as Figure 265 (After L. Cojazzi and G. Guran)

The time required for the cicatrization to reach the end stage of stricture formation is variable. It corresponds to the duration of the esophagitis which averages about one month but may be much shorter or longer. In certain instances it may take several years for the stricture to become pronounced after the original injury, whether it was the result of trauma caused by a foreign body or by chemical burning.

General Characteristics of Strictures

- ✓ **LOCATION** Cicatricial stenosis tends to occur at the levels of the normal, anatomically narrow zones behind the cricoid, near the aorta and the bronchus, and at the lower end. The more or less prolonged physiological arrest which occurs at these levels and the further delay caused by reflex spasm induced by the irritating liquid explain the occurrence of greater injury at these levels than



FIGURE 267 Diagram illustrating the problem produced by the deviations and angulations of the esophagus sometimes seen in cicatricial stenosis of the esophagus. The difficulties and especially the dangers of bougienage under such circumstances can be imagined.

elsewhere. In order of frequency the worst strictures develop first in the broncho-aortic region, then in the region of the mouth of the esophagus, and finally in the lower segment at the level of the hiatus in the diaphragm.

NUMBER Strictures caused by foreign bodies, surgical trauma, external injury, and certain inflammatory lesions are usually single. The strictures resulting from ulcers in the supracardial segment are also single and limited in extent. On the other hand, those which follow chemical burns are most often diffuse, irregular, and multiple. It is of great clinical importance to realize that, in the case of multiple strictures, the lumen of each is not usually in a straight line with those of all the others (Fig. 267).

FORM A single stricture may have a valvular, an annular, or a tubular shape. A valvular stenosis is made up of a cicatricial band caused by contraction of one wall of the esophagus which encroaches upon the lumen to form a partial obstruction.

The annular type is represented by a sort of cicatricial diaphragm with an aperture either in the center or more often on one side (eccentric) (Fig. 269, B, page 383).

In the tubular form, which is the most frequent, the lumen is narrowed throughout the entire involved segment. The passageway through the stenosis is usually tortuous and irregular, ending both above and below in a funnel-shaped transition to normal esophagus. When there are multiple strictures at various levels, one may see tubular stenoses, one superimposed upon the other, or a large part or all of the organ may be involved with extra narrow constrictions at various levels within the disordered segment. In the case of multiple superimposed strictures, the degree of constriction of each tends to increase from above downward.

NEIGHBORING LESIONS With the esophagus, as with all other portions of the digestive tract, the presence of a stenosis induces a certain amount of proximal dilatation. If the diet of the patient is not supervised, a pouchlike

enlargement of this segment may develop above the lesion. This has a tendency to increase progressively to form a reservoir in which remnants of food accumulate, undergo fermentation and thereby produce the lesions of esophagitis. In certain instances this dilatation just above a stricture may give rise to the development of a diverticulum (Fig 270). Figure 282 (page 406) shows an example of a large prestrictural diverticulum which was utilized for an anastomosis with the fundus of the stomach after resecting the damaged esophagus below.

Distal to the stricture the walls of the esophagus become atrophic and the stomach as well.

Finally, the fibrotic process which forms the stricture frequently involves the periesophageal cellular tissue with resultant thickening and adhesions. These tend to fix the esophagus at various points, causing angular deviations or curves which make instrumentation dangerous.

Clinical Characteristics

In the stenoses which follow caustic burns of the esophagus, stricture formation is the inevitable result if the burn has been deep. This is true even if only a few drops have been taken, provided the material becomes thoroughly mixed with the saliva. The stricture develops slowly and insidiously. All cases follow the same general pattern.

After the subsidence of the suffering and anxiety of the days which follow immediately after the ingestion of the corrosive liquid, the patient reassured by the easier swallowing of liquid nourishment and particularly solid food believes that he is cured. This may last several weeks, but sooner or later mechanical dysphagia begins. This dysphagia develops rapidly and is progressive. At first solid foods, later semiliquid foods (purees, etc.) and finally liquids become difficult to swallow. Even water or the patient's saliva becomes arrested in its passage through the upper esophagus.

The dysphagia, furthermore, is permanent with little tendency to fluctuate except when spasm provoked by the esophagitis due to stasis is superimposed. Involvement of the intrinsic nervous system by the scar tissue may be a factor in this aspect of the condition.

The regurgitation of materials ingested is immediate in the case of recent strictures. In chronic cases with a large dilatation of the esophagus proximal to the obstruction the regurgitation is often delayed.

Salivation is abundant, as much because of the accumulation of saliva above the obstacle as because of the activation of the esophagosaltatory reflex which causes excessive secretory activity of the glands. Hypertrophy of these organs, particularly of the parotid glands, has been reported.

The general signs are important from both the point of prognosis and also of therapy. The patient's nutrition is soon affected. Inanition and harrowing thirst make the patient's daily existence miserable and demand the institution of appropriate measures to secure relief. He who has once seen such a patient, whether child or adult, with a pallid expression, an exhausted appearance, drooling at the mouth, eager for even a drop of water, can never forget the picture (Metzianu).



FIGURE 268 Esophagogram of a child with chronic cicatricial stenosis following the ingestion of a solution of barium

Roentgen Examination

The examination should be made at first without giving the patient a barium meal in order to ascertain the condition of the mediastinum. With the ingestion of the opaque medium, information is obtained regarding the nature of the *esophageal stenosis*, particularly its shape and localization. Fluoroscopy demonstrates the length of time required for material to pass through the esophagus as well as the shape of the passageway. It reveals the point of hesitation or arrest and provides information regarding peristalsis and the tendency to regurgitation.

The roentgen films fix the shadows and permit a careful study of the deformities including *angulations* and *diverticula* as well as the establishment of the diagnosis. The shadow of a cicatricial stenosis is characterized by regularity of its contours, no matter how many there may be or what the shape of the stenotic zones may be (Fig 268). The location, the number, and the shape, whether annular, valvular, or tubular are disclosed.

Esophagoscopy

It should be mentioned that the roentgenological findings are not always in agreement with those discovered at endoscopy, particularly with regard to the dimensions of the aperture. This is explained by the rigidity of the walls and the more or less complete abolishment of esophageal peristalsis. When a

cicatricial lesion becomes established, endoscopy shows, beyond the region where the wall is normal or slightly inflamed by esophagitis, raised, flattened, or slightly depressed whitish patches or plaques which indicate the zone of stricture

This stenosis often appears in the form of a fibrous ring of a color more pale than that of the normal mucosa. Sometimes the lumen is in the center. More often the opening greatly reduced in caliber, punctate and eccentrically placed, constitutes the depths of a fibrous funnel in which are star-shaped streaks which have a whitish cast. Such is the appearance of an *annular stricture* which is the most frequent type from the endoscopic point of view (Plate III, 1)

One may also find a cicatricial band crossing the field. This usually has a relatively large lumen with a portion of normal esophageal wall opposite the concavity of the stricture. This is the *valvular type* of stenosis (Fig. 269, A)

Sometimes, also, the esophagoscope impinges upon a thickened rigid, whitish wall. The lumen gradually diminishes in diameter and it is impossible



FIGURE 269 Drawing showing the esophagoscopic appearance of cicatricial stenosis of the esophagus (Eccentric lumen) A Valvular type B annular type

FIGURE 270 Esophagogram of patient who swallowed lye solution with resulting cicatricial stenosis. Film shows proximal dilatation and pharyngo-esophageal diverticulum produced by the increased force exerted by the pharynx in an effort to overcome the obstruction



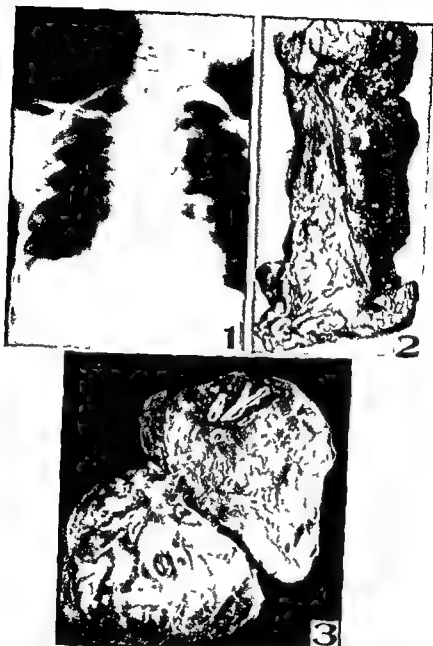


FIGURE 271 Case of severe cicatricial stenosis of the esophagus complicated by bronchiectasis of the right middle and lower lobes the result of pulmonary sepsis induced by aspiration of esophageal contents into the respiratory tract Boy 7 years of age 1, Preoperative appearance showing stenosis and marked proximal dilatation with evidence of collapse of the right lower lobe 2 Specimen of esophagus removed at second operation (esophagectomy) 3 Specimen of lung removed at preliminary lobectomy showing bronchiectatic changes

the case of Metzianu in which a prune pit was gradually forced deeper and deeper into the wall of the esophagus by repeated dilations of the stricture with ultimate perforation and rapid death

Prognosis

The prognosis in cases of stricture is grave both because of the condition itself and because of the dangers of the maneuvers which the treatment requires

In the adult a condition of cachexia may develop in a child the growth and development are retarded, and in both as a result of lowered resistance the susceptibility to certain infections, notably tuberculosis is increased.

The manipulations involved in the exploration and dilatation of a stricture are dangerous. They can propagate the infection of the periesophageal cellular tissues or create a fistula or a pseudodiverticulum by producing a false passage through the wall of the organ. These occurrences are attended by severe symptoms and frequently end in the death of the patient. By constant effort to reduce these complications to a minimum the mortality has been reduced from the earlier figure given by Von Hacker of 40 to 50 per cent to 12 per cent (by Lotheissen), to 5 per cent (by Seiffert), and finally to 2.2 per cent (by Franzas). The tendency to further improvement continues.

From the long range viewpoint, except where the damage is limited, every person afflicted with a stricture of the esophagus has a continuing problem. He leaves the clinic considering himself cured because he swallows food and liquids without difficulty and has gained in weight forgetting the urgent recommendation of his physician that continued surveillance is necessary and that occasional subsequent dilatations may be required.

Statistics based upon series of patients who have had strictures of long duration show an increased mortality. This is especially true with children because of disturbances of growth and with patients who have eventually required a gastrostomy. To quote Chevalier Jackson, "One does not see old persons who have had a gastrostomy tube since infancy." The mortality is much higher in alcoholics, in the tuberculous, and among persons who are beyond middle age. Finally, it must be emphasized again that every dilatation, even after many years and in patients who are followed regularly, carries some risk of a fatal outcome.

Treatment

The treatment of cicatricial stenosis of the esophagus comprises measures intended to prevent stricture formation and measures to overcome a stricture once it has developed.

Prophylactic Treatment

With acute corrosive esophagitis prophylactic treatment involves measures which should be adopted as soon as the condition is recognized (see Chapter 18). Whatever is done at this stage to overcome or prevent the effects of inflammation and trauma has an influence upon the development of subsequent cicatrization and should make it possible by systematic calibration to avoid the formation of an impervious stricture. The use of cortisone is to be noted (see page 370).

When the injury is extensive and deep the esophagus must be put at rest by avoiding all contact with food whether solid or soft. Alkaline solutions, olive oil or neutral powders such as bismuth subcarbonate or kaolin may be administered. The burn of the esophageal wall is an infected wound which must be treated as such and not subjected to the irritation of foreign materials. In severe cases therefore in addition to the treatment of the shock and the employment

of the antibiotics, a gastrostomy must be done early in the course of the treatment. There is everything to be gained by adopting this approach, whereas delay or failure to resort to a gastrostomy leads only to regret. By thus putting the esophagus at rest soon after the injury, the infection can be better controlled and the nutrition can be maintained. The local advantage is that, by keeping the element of infection at a minimum, the sclerosing process is held in check.

In burns of slight or moderate degree the patient may be maintained a few days on rectal or parenteral administration of fluids. Neither the intensity of the dysphagia nor the roentgen examination makes it possible to appreciate the degree of the burn or the extent of the ulceration at this stage. It is only the history of the injury, the degree of infection, and the evolution of the case which make it possible to determine the gravity of the esophageal lesions. All these points have been covered in the chapter on acute corrosive esophagitis (Chapter 18).

Treatment of the Stricture

Treatment of the stricture, once it has become established, must be directed toward the restoration as completely as possible of the normal caliber of the organ. The actual question is relatively simple. Given the sequence of events previously enumerated in the discussion of the etiology, particularly in the case of a burn produced by a caustic, it is important to know if the stricture is dilatable or if it is impervious and not amenable to treatment by this means.

From the practical point of view the patients can be divided into two groups: (1) those who are seen early during the period of acute esophagitis and who are under surveillance from the beginning, and (2) those who are seen late during the period when the stricture is well established or who have negligently abandoned treatment although it was started in the proper manner.

In the first instance, once the injured person is out of danger, the treatment should be begun early in order to safeguard against the development of stenosis or a difficult dilatation. Immediate treatment, on the other hand, has the objection that by leaving a foreign body in the esophagus the healing of the esophagitis may be delayed. The objective is to dilate the esophagus when it is not inflamed and not infected.

The time for intervention cannot be established on the basis of the duration of the process. Certain cases of esophagitis manifest an early tendency to cicatricial contraction (three to six weeks) while others may not show such changes until much later (three to six months). Fluoroscopic examination and the use of roentgen films are not entirely sufficient because they do not make it possible to differentiate between spasm complicating esophagitis or an ulcer and a zone of actual stricture. The only certain means of finding out what the situation may be is to perform an esophagoscopy and to pass a bougie under visual control.

The esophagoscopy can be carried out relatively early. In case of failure, it should be repeated, but if the esophagus bleeds if the bougie impinges upon a zone of inflammation which is difficult to pass, or if the temperature curve becomes elevated, it is better to stop all manipulations and renew the attempt several days later after the esophagus has been put at rest and when the tem-

perature has returned to normal. After two or three trials, another roentgen examination should be performed. An esophagoscopy may then be carried out to recognize the characteristics of the stricture and if possible proceed with careful systematic dilatation.

All these maneuvers, however, should be subordinated to the local conditions and the general state of the patient. If there is no hurry, a gradual dissection of the organ may be accomplished. If, on the other hand, the situation is more pressing, particularly if the state of nutrition and hydration begins to suffer, an emergency gastrostomy must be performed and the final treatment is undertaken only after the patient's general condition has been restored to normal.

Thus the patients may be divided into two additional groups: those who have had a gastrostomy and those who have not. This is of importance in considering the technique which is to be adopted in a given patient.

TECHNIQUE OF TREATMENT AMONG PATIENTS WHO HAVE A GASTROSTOMY
The usual Haslinger instrument or one of its modifications is recommended for this purpose. After preliminary sedation, local anesthesia is produced by the topical application of pontocaine (1 per cent solution). In very young or uncooperative children, general anesthesia is advisable.

The esophagoscope introduced according to the accepted technique is passed through the mouth of the esophagus and is gradually pushed deeper under visual control. Blood and pus are evacuated with the aspirator. In case of continuous oozing of blood, pledgets of cotton soaked in a solution of cocaine and Adrenalin are applied. As the region of the stricture is approached, a search is made for the lumen through it. The aperture is often of pinpoint size and usually eccentrically located (Fig. 269, page 383). It may be recognized sometimes only by the escape from it of saliva or secretions.

Penetration of the opening is accomplished with the aid of pliable radiopaque bougies. It is well to carry out the procedure under the guidance of both endoscopic visualization and fluoroscopic control. As the bougie is pushed further, the roentgenologist watches its passage in the esophageal lumen and gives warning immediately if it curls up above the stenosis. The progress of the bougie is followed until it reaches the stomach.

From this point on, the following steps may be followed. With the aid of the fluoroscope, the extremity of the bougie may be grasped with a hemostatic forceps introduced into the stomach through the gastrostomy stoma and drawn to the exterior. Various other methods of grasping the gastric end of the bougie have been devised. These include, especially in children, the use of retrograde esophagoscopy, a thread furnished with a lead shot, a radiopaque ureteral catheter to be extracted with the aid of fluoroscopic visualization using a bullet forceps, the employment of a cystoscope or urethroscope equipped with a catheter having a bent tip and the use of the umbrella technique. With the latter, several black threads are attached to the distal extremity of the bougie before it is introduced. These are folded back along the bougie and trail along with it through the stricture. On arriving in the stomach, which has been filled previously with water, these threads unfold and with the assistance of a nasal speculum introduced through the gastrostomy stoma, one of the threads is

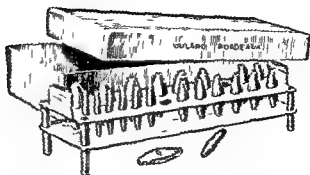


FIGURE 272 Battery of dilators for dilatation of the esophagus on an endless thread

grasped with a forceps. Traction on the thread brings the tip of the bougie within reach.

A long strand of No. 6 silk thread is attached securely to the upper extremity of the bougie, which is then drawn out of the esophagus through the gastrostomy opening. The oral and gastric ends of the thread are tied together, creating a circle of thread which can be used for the dilatation. This is carried out by means of olive-shaped metallic bulbs calibrated according to the Charrière scale which are attached to the silk thread (Figs. 272 and 273).

A start is made with the smallest sizes. Three of the olives are attached to the string in succession with the smallest size, which is to pass through the stricture first, placed lowermost. The passage of these three dilators with the occasional assistance of the fluoroscope requires a variable period of time from several hours to several days. Usually a period of from two to three days is necessary. During this time the patient is kept quiet, antispasmodics are administered, and, in case of pain, morphine is given hypodermically.

The sessions of dilatation are repeated every week or every fortnight according to the length of time required for the passage of the olives through the esophagus as verified by fluoroscopic examination or according to marks made by tying knots in the thread to indicate the different stages of the circuit. The first knot indicates the distance between the dental arch and the first stricture. When this knot is at the dental arch, the first olivary dilator is in contact with the first stricture. A second knot marks the length of the esophagus as far as the last olive. When this knot is in contact with the dental arch, the last olive has passed through the cardia. After the succession of dilators has been pulled through the gastrostomy opening, the thread is changed.

Accidents which may occur include, first, *blockage of the dilators*. This may be because they turn over. This occurrence may be avoided by making a rather large knot between each pair of dilators and placing the olives very close to each other.

Another technical difficulty is *breakage of the thread* because of injury on the teeth or because it is weakened by the digestive action of gastric juices. To avoid the latter eventuality, it is recommended that the thread be kept moving around the circuit and that it be changed frequently during the interval between dilatations. Every fourth day is a good routine to follow.

A final complication may be the *development of esophagospasm*. In the course of the first few sittings, especially in a nervous patient, long and painful attacks

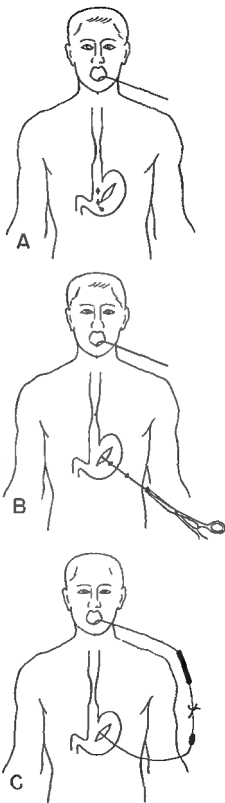


FIGURE 273 The procedure using a thread with lead shot *A* The lead shot on the end of the thread has reached the stomach *B* A forceps introduced through the gastrostomy stoma seizes the lead shot under the guidance of the fluoroscope and draws it outside the gastrostomy stoma *C* The orogastric circuit is established and on it olivary dilators or sounds are introduced

of spasm may be encountered. These are to be overcome by absolute rest and the administration of sedatives combined with antispasmodics.

Figures 274 and 275 illustrate a modification of the method whereby a rubber or plastic tube is drawn into place within the stricture and allowed to remain for several days or weeks at a time. Larger tubes may be inserted from time to time as the diameter of the stricture increases. This has the advantage that, while aiding and maintaining the enlargement of the passageway, a certain amount of liquid nourishment may be consumed by the patient with the tube in place. With it also the effects of esophagospasm are overcome.

Duration of Treatment The procedure of dilatation by an endless thread should be pursued for a long period of time. The average duration is from eighteen months to two years. The olives should continue to be passed in larger and larger sizes until their passage can be carried out without any difficulty. The thread is then allowed to remain in position after the termination of the period of dilatation in case it should be needed subsequently. After a trial period of two or three months the thread is removed, but the gastrostomy stoma is retained until a normal peroral intake of food has been resumed.

Alimentation Because of the abnormal circumstances of their condition, special attention must be paid to the method of feeding these patients. Although the intake of food must be restricted during the period of esophagitis, the deglutition of nourishing liquids, semisolids, and finally solids during the period of cicatrization helps to bring about a gradual dilatation during the interval between the treatments. During the first few days of the dilatation the patient is allowed thin liquids. After a few days he may take milk, broth, and diluted cooked cereals. Later he may have thick soups, pureed vegetables, and finally bread and chopped meat. During the actual passage of the dilators, on the other hand, all food must be withheld.

TECHNIQUE OF TREATMENT AMONG PATIENTS WHO DO NOT HAVE A GASTROSTOMY The aperture through the stricture must be sought and a small bougie inserted under direct vision using the esophagoscope. Much patience is required. The illumination must be good. Repeated aspiration is necessary. The application of pledgets of cotton or gauze soaked in a vasoconstrictive liquid is helpful. The eye must scrutinize every fold and zone of granulation, watching for secretions or bubbles of air. One must palpate cautiously with the tip of the bougie. Finally, the orifice of the stricture is discovered and the bougie is made to enter. The sense of touch is extremely useful in this maneuver.

If the filiform bougie goes in without difficulty and if it is not held up by irregularities in the wall of the esophagus, a larger bougie may be introduced, but *never by the use of force*. As soon as the passage becomes difficult, the attempt must be given up. If this rule is not followed, the next day will find the insertion of the bougie impossible. For this reason if the introduction of the filiform bougie is difficult it should be left in place for two hours or more. This practice often accomplishes more than a dilatation which is too rapidly pursued. The technique of bougienage from this point on has already been described in Chapter 18.

To avoid harm and to produce the best results, the dilatation must be conducted with patience, gently and easily. The seances may be held at daily

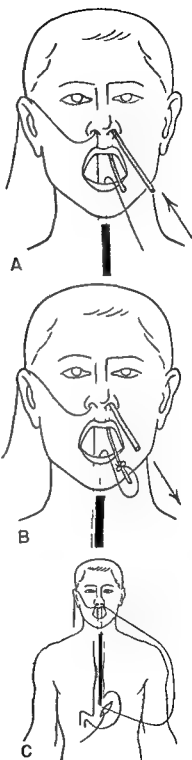


FIGURE 274 Modification of the continuous thread technique *A* Passage of the thread on which a tube dilator has been attached through the nose with the assistance of a Nelaton catheter *B* The thread of the dilator tube is tied onto the catheter and drawn through the nose *C* The orogastric circuit and dilator in usual position The caliber of the dilator is increased progressively

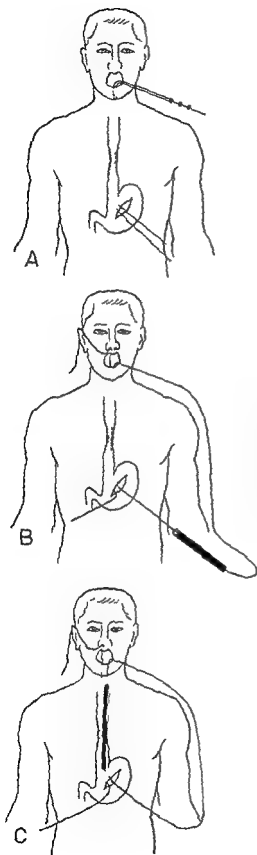


FIGURE 275 Further modification of the thread with leaded shot technique
A The orogastric circuit has been established with a double thread *B*, One thread passes through the nose this is an accessory thread to be used in case of breakage of the other thread to which a tube dilator is attached *C* Dilator in position within the stenotic segment (After David [Galatz])

intervals or less often according to the reaction and the progress of the stretching. At each treatment two or three successive sizes may be passed but always *without force, without causing pain and without giving rise to an elevation of temperature*. Pain, fever, and bleeding are indications for a cessation of effort until the condition improves.

Once the lumen has been established it is helpful and safer to encourage the patient to swallow a silk thread in order to maintain access to the passageway. Once the end of the thread has reached the intestinal tract, it becomes fixed so that it cannot be withdrawn. From then on the thread serves as a guide for the passage of bougies of increasing sizes (see Chapter 18). In adults the dilatation should be carried at least to size 26 or 28 and in children to 15 or 18.

If the dilatation does not succeed, if it produces pain or fever, or if there is more than one stricture so that dilatation cannot be carried out all the way down the esophagus, a gastrostomy should be performed. After the subsidence of esophagitis and spasm as a result of putting the esophagus at rest or of keeping the portion proximal to the stricture clean, the dilatation may be resumed using the continuous thread technique.

With multiple stenoses each must be dilated in turn.

Management of Special Problems

In some instances the *stricture can be passed only from below upward*. This demands the employment of retrograde bougienage. The various methods of accomplishing this have been described (Chapter 4). Once a bougie has been passed all the way to the mouth, a thread may be attached, the instrument withdrawn, and the two ends of the thread tied together for use as described above.

Under certain circumstances the *stricture appears to be impervious*. Although almost everyone agrees that an opening can usually be found, sometimes all methods fail when, especially after severe caustic burns, there exist several strictures, when the walls of the esophagus are fused together along the entire length of the strictured segment or when the cardiac orifice has disappeared and been transformed into a mass of cicatrix. Heindle found six cases of impermeability in thirty-seven patients. In this situation the patient is condemned to live with a gastrostomy unless a substitute esophagus is constructed by surgical means (see Chapter 20).

So long as an opening can be preserved, however, and barium can be observed to pass into the stomach, there is reason to be hopeful. Bougienage either direct or retrograde may one day succeed in overcoming the difficulty so long as patience and perseverance are maintained.

It is absolutely essential to make certain that the stricture is actually impermeable and that one is not dealing with a transitory stenosis. In practice there are extreme variations from open permeability to complete occlusion depending upon the presence or absence of esophagitis and spasm. In a suspected case, after prolonged rest of the esophagus, it may be helpful to watch the descent of a swallow of iodized oil under the fluoroscopic screen. Sometimes the instillation of a colored liquid (methylene blue or indigo carmine) at the upper end of the stricture may lead to the conclusion that the lumen is not

completely obstructed when some of the color appears in gastric contents aspirated through the gastrostomy stoma. The roentgen examination, however, is often unreliable, but when not even a threadlike shadow indicating the course of the esophageal lumen can be discerned, the chances of success with bougienage by any method are exceedingly slight.

Very rarely, if the stricture is sharply localized in extent to not over 1 cm in length along the esophagus, it may be penetrated by surgical intervention through the esophagoscope with a biting forceps or other instrument. This must not be attempted without the indispensable assistance of the roentgenologist to check the position of the instrument in relation to the stricture. The diathermy knife has been used in a few instances for this purpose, but this method is not without its dangers.

With the latter technique it is necessary to proceed cautiously under the guidance of the fluoroscope. First the superior surface of the stricture may be attacked and then the inferior aspect by retrograde esophagoscopy. An opaque bougie is left in the lumen of the esophagus as a guide. When several millimeters of depth have been achieved, the bougie is thrust into the lumen and left in place for several days until the electrocoagulation is tried again. If several attempts to establish a passageway are ineffective, the method should be abandoned to avoid disaster.

CHAPTER 20

Esophageal Substitution

General Principles

When dealing with a bleeding penetrating, or painful ulcer, with an esophageal fistula or with a carcinoma, it is of course essential to remove the lesion by performing an esophagectomy the magnitude of which depends upon the circumstances. With cicatricial stenosis however, it is not always essential to remove the diseased segment. In fact with caustic burns the periesophageal inflammatory reaction and fixation of the esophagus make the dissection to free the organ exceptionally difficult. Furthermore, it is not always possible to determine with ease the upper extent of the injury so that the choice of the level at which the esophagus must be divided may be troublesome. These difficulties however, are by no means insurmountable and the decision rests with the personal preference and technical skill of the surgeon.

In the cervical region where a skin tube is employed, a resection of the lesion is always performed.

Methods

The replacement of diseased segments of the esophagus either after resection or as a sidetracking procedure may be accomplished by one of three methods: (1) the use of a tube of skin, (2) the interposition or substitution of another portion of the alimentary canal, or (3) the use of a prosthesis of foreign material.

EMPLOYMENT OF A SKIN TUBE

Except in the cervical segment the employment of a skin tube to replace a segment of the esophagus is cumbersome, time-consuming, and functionally unsatisfactory. The procedure must be performed in multiple stages which may require many months for their completion. If the skin is hairy, obstruction from the accumulation of hair inside the tube becomes a problem. Stricture formation

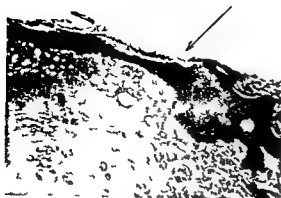


FIGURE 276 Photomicrograph showing healing of the skin to the esophagus. Note particularly the accurate first intention union of the esophageal and cutaneous epithelial layers.

also causes difficulty, especially at the point where the skin tube is connected either to the stomach or to the intestine in cases in which partial interposition of a segment of bowel is used. In fact, the only circumstance where the skin can be relied upon to heal to the mucous membrane of the alimentary tract without the formation of a stricture is when it is united either to the pharynx or to the esophagus. This is because the union of the two layers, because of their structural similarity, takes place by primary or first intention healing whereas that which occurs between the skin and the glandular mucous membranes of the stomach, the jejunum, or the colon takes place by second intention with granulation and subsequent cicatricial contraction. Figure 276 illustrates the kindly healing of the dermal epithelium to the mucosa of the esophagus. Because of this fact, the skin may be used with impunity to replace the cervical esophagus.

Cervical Skin Tube

Various modifications of technique are available for the construction of a short tube from the skin of the neck with which to replace the cervical esophagus. With rare exceptions the need for this procedure is confined to the operation for the removal of a small carcinoma in this segment. For this reason the technique is described in Chapter 28 which deals with the surgical treatment of carcinoma and, in order to avoid repetition, is not given here.

Antethoracic Skin Tube

In one instance only is it feasible to use a skin tube to replace the thoracic esophagus. This is the unusual circumstance where a portion of the distal esophagus can be preserved with which to attach the lower end of the tube. To accomplish this the stomach and lower esophageal stump must be transplanted to the subcutaneous space of the lower anterior chest wall. The integrity of the anastomotic vascular channels in the fundus of the stomach and particularly along the lesser curvature must be preserved in order to avoid necrosis of the remnant of lower esophagus which under these conditions depends for its nourishment upon the gastric blood supply.

Figure 277 illustrates a successful case of this sort in a man who has survived seventeen years without suffering any nutritional disturbance or obstructive difficulties following the performance of a Thorek operation for carcinoma



FIGURE 277 Photograph of the chest of a patient who had an esophagectomy by the Thorek method followed by skin tube restoration of continuity (17 year survival)



FIGURE 278 Roentgen film of patient shown in Figure 277 showing the skin tube filled with barium

Figure 278 shows the appearance of the tube when filled with barium. This patient, however, in common with all the others, finds it necessary after a few mouthfuls have been swallowed to push the food which accumulates in the skin tube down into the stomach with a sweep of the hand.

EMPLOYMENT OF OTHER VISCERA OF THE ALIMENTARY TRACT

With the exceptions noted above, the only satisfactory way to restore continuity after the loss of all or a portion of the esophagus is to substitute some other portion of the digestive canal. This may be done after a segment of the esophagus has been removed or as a by-passing procedure, leaving the esophagus in position. The former should always be done when possible in the treatment of carcinoma, ulcers, and fistulae. The latter may be preferable in benign strictures and as a method of palliation in cases of carcinoma where the tumor cannot be removed.

An important technical consideration when a by-passing procedure is being employed is whether or not the esophagus should be transected above the lesion. The use of a lateral anastomosis leaving the esophagus intact, as recommended by Yudin for jejunal substitution, has the advantage of simplicity. A serious disadvantage, however, is that a dependent blind pocket of esophagus is left below the anastomotic level in which food may stagnate to produce irritation with esophagitis or even acute infection. It is preferable, therefore, to divide the esophagus at an appropriate level. The distal end is then closed and inverted and the proximal end is utilized for the anastomosis, thus avoiding stagnation by delivering all ingested material directly into the transplanted organ.

According to the circumstances in each case and depending often upon the personal preference of the surgeon, the transplanted viscus or segment used for the substitution may be brought up to meet the proximal end of the esophagus in one of several ways. In every instance, however, it is imperative to maintain an adequate blood supply for the organ employed.

THE SUBCUTANEOUS ROUTE In some ways the easiest and safest measure is to carry the fully mobilized organ or segment up to or close to the neck through a subcutaneous channel. This was the method first successfully employed by Roux when he used a segment of jejunum. The subcutaneous tissues have the advantage of ready accessibility so that in case of the failure of blood supply or a leakage from the anastomosis, a major calamity is avoided by not having the accident occur within the thoracic cavity. The chief disadvantage is that there is disfigurement because of the obvious abnormal presence of a portion of hollow viscus beneath the skin. This objection has significance particularly with women and sensitive children.

THE MEDIASTINAL ROUTE An approach to the problem which has gained favor in recent years is to use the anterior mediastinum. This is particularly useful when a portion of intestine is being drawn up, but if the stomach is employed, it may be difficult to find room at the base of the neck. This method is technically easier and less likely to be followed by pulmonary and cardiac complications than when a lateral thoracotomy is employed. The space may be prepared to receive the transplanted viscus either by blunt dissection from above and below (Fig. 279) or by performing a midline sternotomy.

Whenever the esophagus has been removed, however, it is preferable to use the posterior mediastinum which has already been prepared for the receipt of the transplant by the dissection necessary to carry out the resection.

The employment of either the anterior or the posterior mediastinal route,

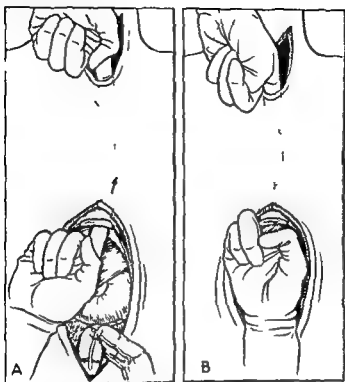


FIGURE 279 Drawing to show technique of dissection of the anterior mediastinal space to receive the transplanted colon (or other viscus) A Start of dissection with fingers from above and below B Dissection almost complete (Adapted from J. Perrotin)

however, makes exacting demands upon the technical skill of the surgeon. Any failure of blood supply or the development of an anastomotic leak may lead to a fatal outcome and *must be avoided without fail*.

THE TRANSPLEURAL ROUTE The earliest experiences with esophagectomy followed by immediate restoration of visceral continuity involved the use of the pleural cavity through which to pass the transplanted viscus. The employment of this method has been attended by hundreds of successes and cannot be lightly abandoned. It is subject especially when the left side is used to the disadvantage that the stomach or intestinal segment is allowed to remain in a position which interferes to some extent with the functioning of the lung. This mechanical objection is important only during the early convalescent period when difficulties in raising secretions tend to favor atelectasis of the lung on the side which has been used. Ultimately, however, the patient suffers no inconvenience from the presence of the transplanted organ within the pleural cavity.

For this reason nowadays the posterior mediastinal route is more often employed with the possible exception of the instance where the anastomosis must be performed in the neck.

Employment of the Stomach for Esophageal Replacement

On first thought it seems to be mechanically ideal to replace portions of the thoracic esophagus after excision or by-passing by means of the stomach. The stomach can be mobilized successfully to such an extent that an esophago-

DISEASES OF THE ESOPHAGUS

gastric anastomosis can be performed at any level within the thorax and even in the neck or at the pharynx if so desired. This procedure can be accomplished with a smaller risk of leakage and other complications resulting from technical faults than the use of the colon and to a lesser degree the jejunum. The formation of a stricture at the anastomosis and leakage leading to fistula formation are avoidable technical failures.

On the other hand, it must be acknowledged that there are physiological disturbances following this procedure which may mar the result to some extent in susceptible subjects, especially persons whose operation was performed for the purpose of resecting a peptic ulcer, a certain amount of esophagitis may develop. On rare occasions this may occur soon after the operation. More often it is a late complication. The important role of delay in the emptying of the stomach in these patients has been discussed in Chapter 15.

An occasional complication which may depend also, upon an altered physiology is the postoperative development of a gastric ulcer. This may occur during the early recovery period before the patient leaves the hospital. These ulcers are always painless and may reach the stage of perforation rapidly without the patient being aware that anything is wrong. Deaths from perforation into the mediastinum or from erosion of the thoracic aorta have been observed.

Remote difficulties involving failure to maintain an adequate nutritional state, the development of anemia, and the frequent occurrence of the post-gastrectomy so-called 'dumping syndrome' are conditions which can be prevented or at least minimized if the patient is fortunate enough to be attended by an interested physician. It should be emphasized that patients after this as well as after all other gastric operations, continue for many years to require specialized medical care and supervision.

In general, therefore, if certain conditions are met, the stomach may be used with satisfaction whenever an esophagectomy involving an intrathoracic anastomosis is performed. In patients who are to be treated by esophageal side tracking, although the stomach can be used for the purpose, it is preferable to employ either the jejunum if the anastomosis is to be in the mediastinum or the right colon if it must be made in the neck.

To avoid duplication, the technical details of utilization of the stomach to replace the esophagus after esophagectomy, which are described in full in the chapter on the treatment of carcinoma (Chapter 28), are not repeated here. When dealing with a carcinoma, special emphasis must be placed on radical extirpation including a large segment of the esophagus and all lymph nodes which are accessible to the surgeon. It should be borne in mind, however, that whereas a radical policy is not only justifiable but also highly desirable when dealing with a fatal disease such as carcinoma the same attitude may not be applicable in the case of benign lesions.

EMPLOYMENT OF THE STOMACH FOR ESOPHAGEAL REPLACEMENT IN THE TREATMENT OF BENIGN LESIONS Almost without exception the only types of benign inflammatory stricture of the esophagus which may require treatment by resection are those due to stenosis resulting from esophagitis with ulcerations and those resulting from chemical burns induced by the ingestion of sclerosing caustic solutions.

Benign Stricture and Peptic Ulceration The characteristic syndrome consisting of an underlying congenital anomaly with a short esophagus and hiatus hernia, the thoracic portion of the stomach resembling an inverted cone, has been described. Superimposed upon this anatomic arrangement there is often esophagitis of the lower end just above the cardia and gastritis of the thoracic portion of the stomach. This leads to ulcerations of the peptic variety and ultimately to cicatrization and stenosis. There is also a high incidence of coexisting duodenal ulcer in this condition, sometimes also with stenosis. This fact must be kept in mind when a resection for stricture at the cardia is being considered. Separate surgical treatment for such a duodenal stenosis may be necessary before the proximal stricture is resected. Sometimes both conditions can be relieved at the same operation with a pyloroplasty to correct the former and a resection or an esophagoplasty to overcome the latter (Chapter 15).

During the dissection required to free the lower esophagus and upper stomach in cases of this sort, it will be observed that the surrounding tissues of the mediastinum are exceedingly adherent, making the development of planes of cleavage more difficult than in the average case of noninvasive carcinoma. Often most of the dissection must be accomplished with sharp instruments. When the time arrives for the transection of the esophagus it is frequently difficult to determine the level where the pathologic changes merge with the normal esophagus. This is a matter of great importance, however, because it has been found with esophagitis that if the esophagus is transected too close to the diseased area there is a decided tendency for the inflammatory process to spread proximal to the anastomosis.

Another important consideration in dealing with this disease is that it is complicated in a significant number of cases by the development of carcinoma. This is apparent from the observation that approximately 15 per cent of cases of carcinoma in the region of the cardia and lower end of the esophagus develop in the presence of a short esophagus and hiatus hernia. Therefore the surgeon should search for possible evidences of malignant change during the operation and plan the extent of his resection accordingly.

Because the condition is actually a manifestation of the ulcer-forming tendency in patients who happen to have the anatomic arrangement which favors the regurgitation into the esophagus of acid and pepsin in the gastric juice it is important to diminish as much as possible the production of this secretion. An effort must be made to make the resection as effective in this respect as the distal partial gastrectomy (subtotal) which has come to be the standard procedure (exclusive of vagotomy) in the treatment of ulcer of the duodenum. This result may be accomplished by adopting the most radical resection of the stomach and diseased esophagus which can be carried out without interfering with the possibility of restoring continuity by esophagogastronomy. The procedure which approaches as nearly as possible the ideal of radical gastrectomy for ulcer in cases of this sort has been mentioned in Chapter 15 (Fig. 238). By this means the majority of the lesser curvature and all of the body and fundus of the stomach are removed, leaving only a tube of greater curvature with which to perform an end-to-end anastomosis with the esophagus. Once the dissection has been completed and the mobilization of the stomach has been

DISEASES OF THE ESOPHAGUS

accomplished, the remainder of the operation proceeds in the manner to be described in Chapter 28, where an end to-end anastomosis is employed. In the majority of these patients, because of the hiatus hernia the left gastric vessels lie much closer to the diaphragm than in a patient with a normal anatomic arrangement. Their division is thus easily accomplished.

Because it is necessary to resect a large segment of both esophagus and stomach, the continuity of both vagus nerves must be interrupted for technical reasons. The effect of vagotomy may be very troublesome in these patients because of the reduction in gastric peristaltic activity and the hypertonicity of the pylorus. This tendency to delayed emptying of the stomach can be overcome to some extent, at least in the absence of organic stenosis at the pylorus or duodenum, by dividing the gastrocolic and gastrohepatic ligaments as far as the region of the pylorus in exactly the same manner as required whenever an unusually high intrathoracic anastomosis is to be performed. This has the effect of interrupting some of the sympathetic nerve fibers whose activity might otherwise produce excessive tonicity of the pylorus. In cicatricial stenosis of the pylorus obviously something more is required and the most effective procedure is a Heineke-Mikulicz pyloroplasty. This can always be done through the thoracic incision, although enlargement into the abdomen may be necessary occasionally to gain sufficient exposure.

Cicatricial Stenosis from Chemical Burns Cicatricial stenosis from chemical burns of the esophagus presents similar difficulties which depend upon the nature and extent of the lesion. In this condition likewise there is usually marked evidence of periesophagitis and inflammatory fixation, making the dissection difficult. In fact, the freeing of such an esophagus is sometimes more difficult to accomplish than in any other type of disease in which a resection is required. It is difficult also to determine where the proximal dividing line between the normal and the diseased portions of the esophagus lies. This is almost invariably higher than one might guess by inspection and even by palpation. Reference to the roentgen film showing the pattern produced by the ingested barium mixture may be necessary in order to determine the correct level of transection. It should be remembered also that the proximal margin of the cicatrized area is almost invariably funnel shaped. On the other hand it is not necessary to cut across the esophagus any higher than the last normal tissue which lies just proximal to the lesion. This is of great importance because of the necessity of conserving as much proximal length as possible when the injury is extensive. It is important to remember also that in some patients with a stricture resulting from ingested corrosive liquids, there is in addition to the esophageal injury a chemical burn of the pyloric end of the stomach. This is usually sharply localized at the pylorus but may occasionally involve some of the antral portion of the stomach as well. In a few such patients, although the ability to swallow may be temporarily maintained by means of bougienage, the inaccessibility of the pylorus allows the cicatrization to progress to the point of stenosis and ultimate obstruction. The surgical relief of the pyloric stenosis should be accomplished if possible by some procedure which will produce a minimum of shortening in order to preserve sufficient length and flexibility of the stomach for use



FIGURE 280 Roentgen films of a patient age 17 at the time of operation for the relief of a lye stricture of the esophagus *A* Preoperative esophagogram almost complete occlusion due to cicatricial stenosis of the lower third *B* Film taken 5 years later showing the stomach in the chest and anastomosis at the arrow mark

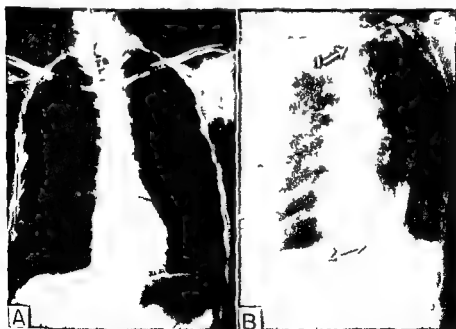


FIGURE 281 Roentgen film of a patient age 22 who was operated upon for cicatricial obstruction of almost the entire esophagus resulting from ingestion of lye with suicidal intent *A* Preoperative esophagogram (Note small pharyngo-esophageal diverticulum already present) *B* Postoperative film following subtotal esophagectomy and esophagogastric anastomosis 1 anastomosis 2 pylorus



FIGURE 282 Roentgenogram showing the use of a large pharyngo esophageal diverticulum for an anastomosis with the stomach after subtotal esophagectomy for a cicatricial stenosis occurring at the age of 2 years. Operation performed at age 22 years. Arrow points to the anastomosis. Pylorus shown at P (No subsequent difficulties 10 years later)

later should an esophagectomy become necessary. The Heineke-Mikulicz pyloroplasty serves admirably for this purpose.

Ordinarily it is possible, even after gastrostomy, has been done to mobilize the stomach sufficiently to carry out the usual procedure as described above with a partial or even subtotal esophagectomy and a primary esophago-gastric anastomosis within the left thoracic cavity or actually within the neck. However, in those patients who have sustained an injury to the major portion or all of the esophagus and who have also developed a stricture of the pylorus which requires surgical relief with resultant foreshortening of the stomach it may be necessary to use the jejunum or the colon for the restoration of continuity.

An example of the use of the stomach to replace the lower esophagus after extensive cicatrization resulting from a chemical burn is shown in Figure 280, A, B. That the entire stomach may be mobilized to replace the esophagus after nearly total destruction of that organ as a result of chemical injury is shown in Figure 281, A, B. Figure 282 provides another example of replacement by the stomach. In this instance a large pharyngo-esophageal diverticulum which had formed proximal to the upper limits of the stricture in the cervical esophagus was employed for the anastomosis (diverticulogastrostomy).

Employment of the Jejunum for Esophageal Replacement

The first successful case of visceral substitution to replace an injured esophagus is that reported by Roux of Lausanne in 1907. The patient was a child

who had swallowed a caustic liquid, thereby sustaining a severe chemical burn of the lower esophagus which resulted in an impervious stricture. The antethoracic subcutaneous route was employed. The lower end of the isolated jejunal transplant was anastomosed end to-side with the stomach. The patient survived many years.

The advantages of using the jejunum are that access to it is relatively easy and that bacterial contamination is more readily avoided than with the colon. For physiological reasons it is preferable to the stomach as a substitute because, like with the colon, the stomach need not be disturbed and the vagus nerves are therefore not severed.

When used, however, the lower end of the segment must be anastomosed with the stomach as in Roux's original case. If this is not done the food does not enter the stomach which then becomes more like an accessory organ of the gastrointestinal tract and its food-storing function is lost. The patient who is left in this condition is subject to the discomforts of those who are fed through a jejunostomy where the food enters the jejunum beyond the stomach. Failure of absorption, dumping symptoms, and diarrhea are likely to be the result. The loop should be brought up in an isoperistaltic direction because, if it should be reversed, a disastrous physiological obstruction is the result. The method should be used in cases of impervious lye stricture or occasionally to by-pass a carcinoma which cannot be removed.

It should be noted, however, that the technical problem of preserving the blood supply of the transposed segment is exceptionally difficult although the jejunum lends itself readily to passage through a subcutaneous tunnel or through the anterior mediastinum. The posterior mediastinal space is available if an esophagectomy has been carried out. The pleural cavity can be used in the latter condition also.

TECHNIQUE OF JEJUNAL BY-PASS (ONE STAGE) A longitudinal abdominal incision is made through the rectus sheath close to the midline. The proximal jejunum is identified and its mesenteric vessels examined. Through an incision in the mesenteric peritoneum several of the upper jejunal arteries and veins are divided close to their origins, preserving the primary and secondary anastomotic vascular arches (Fig. 283). Because of the relative lack of intercommunicating channels in the bowel just distal to the ligament of Treitz, this portion must not be included in the isolated loop (Fig. 284 B). By removing the peritoneum from each side of the mesentery of the loop which is to be used, the curve in the bowel can be straightened to a considerable degree.

The bowel is then divided between clamps or ligatures beyond the first few inches and a trial of length is made by pulling the partially mobilized distal end out of the incision and up over the anterior chest wall. If not enough length is available, one or more jejunal arteries must be divided. Finally when the proper amount of mobilization has been accomplished, the loop is replaced within the abdomen and the anterior mediastinum is prepared for its passage.

Two methods are available to accomplish this. The first consists in enlarging the abdominal incision upward to the base of the neck, using a sternotomy chisel to cleave the sternum. This is quickly done and has the advantage that tearing and injury to the blood supply of the transplanted jejunal loop is avoided.

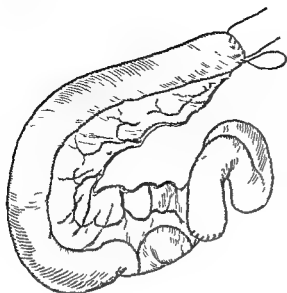


FIGURE 283 Drawing showing the result of proper mobilization of jejunal segment preserving vascular continuity by way of the arcades in the mesentery.

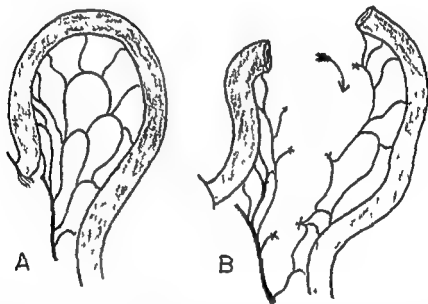


FIGURE 284 Diagram to illustrate the principles involved in the preservation of the jejunal blood supply. *A* Note end artery arrangement of the arteries nearest the ligament of Treitz. *B*, correct site for division of the mesentery, avoiding the proximal segment where the arterial arrangement is unfavorable.

The bowel can be laid in place gently, without manipulation or traction and with an absolute minimum of trauma.

The second method, which is more commonly used but which is not so satisfactory, consists in the creating of a space beneath the sternum by blunt dissection using the scissors and fingers. Through a cervical incision parallel with the anterior border of the left sternocleidomastoid muscle the deep fascial space in the neck is entered and the upper portion of the dissection is carried out taking pains to stay close to the posterior surface of the sternum. When as much as possible has been accomplished in this manner, the anterior mediastinum is entered from below by cutting the innermost fibers of the diaphragm

on each side of the xiphoid process, and the dissection is carried upward in the same manner until the cervical area is reached (Fig 279)

Through the upper end of the thoracic extension or through the short cervical incision if the second method is employed, the cervical esophagus is mobilized and transected. The distal end is closed and inverted with Lembert sutures. The proximal end is drawn out for the performance of the anastomosis. After the necessary length of bowel has been prepared the distal portion is pulled up through a short incision in the transverse mesocolon to a point as high as necessary in the chest. The edges of the opening in the mesocolon are sutured to the bowel where it passes through it (Fig 286)

The supposition exists among surgeons that, once the esophagus has been divided, the anastomosis must be made end to end. This can be accomplished readily when only a short segment of jejunum is needed to reach the lower or middle esophagus. When a longer segment is required however, it is impossible in many instances to avoid arching of the distal end even after some of the secondary vascular arches have been divided. When an effort has been made to overcome this awkward situation by dividing a few of the terminal arterial branches, the blood supply of the end of the bowel immediately becomes inadequate for use in the anastomosis (Fig 285). To avoid this dangerous eventuality, the end of the bowel should be closed and the anastomosis should be made between the end of the esophagus and the side of the antemesenteric surface of the arching end. Two layers of fine silk sutures are employed

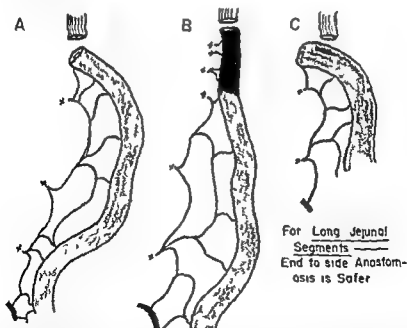


FIGURE 285 Diagram illustrating the technical problem presented by the use of a long jejunal segment. Reason for making the anastomosis end to side. A Unavoidable curving of the end of the jejunal segment due to the effect of the vascular arcade. B Danger to the blood supply of the terminal 2 or 3 inches if enough vessels are divided to straighten the end for an end-to-end anastomosis. Black area indicates the length of bowel which is certain to become necrotic. C Difficulty avoided by closure of the end of the jejunum and use of end-to-side anastomosis.

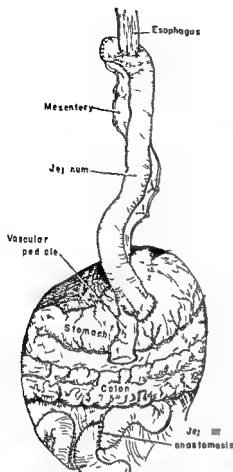


FIGURE 286 Diagram to show the anatomical arrangement after completion of the operation of jejunum replacement of the esophagus in one stage. Note end-to-side esophagojejunal anastomosis, distal end-to-side jejuno gastric anastomosis, vascular pedicle (and jejunal segment) brought up through the transverse mesocolon, end-to-end jejunal anastomosis after isolation of the transplanted segment.



FIGURE 287 Roentgenogram of patient age 22 operated upon for cicatricial stenosis and ulceration of the entire esophagus save for the proximal 1 inch (accidental ingestion of 1% solution). A Preoperative esophagogram. B Film showing transplanted long jejunal segment brought up through the left pleural cavity. Note end-to-side esophagojejunal anastomosis (arrow).

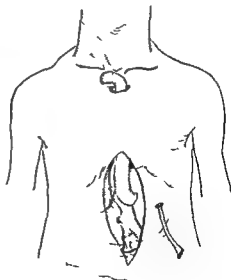
The next step is to sever the transplanted jejunum at an appropriate point close to where it lies near the stomach. In doing this no mesenteric vessels except a few terminal branches as they enter the bowel wall are divided. If this precaution is not observed, the blood supply to the entire segment may be compromised. The lower end of the segment thus isolated is anastomosed to the anterior wall of the stomach as near as possible to the fundus. If, as is so frequently the case, there is a gastrostomy, the stomach must first be freed from the abdominal wall and the stoma closed with interrupted Lembert sutures. Finally, the remaining end of jejunum is anastomosed end to end with the free end below the ligament of Treitz (Fig. 286).

The incisions are closed in the usual manner. If the sternum has been divided, the edges must be held together with several steel wire sutures passed through the body of the bone in order to insure stability. The mediastinum should not be drained. Figure 287 shows an example of the use of the jejunum for total esophageal replacement.

Aftercare The aftercare does not present any exceptional difficulty. Oral intake must be restricted, but on the first postoperative day water may be allowed in amounts equal to 30 cc per hour. From then on the intake is advanced gradually according to well accepted principles. If ordinary caution is observed, leakage from the suture lines need not be feared. Failure to heal is caused almost without exception by inadequacy of the blood supply on the jejunal side due usually to faulty technique. Parenteral administration of water and electrolytes with vitamins added is continued until the oral intake is adequate for the patient's needs. The patient is allowed out of bed by the second day. Other measures are instituted as indications arise.

TWO STAGE TECHNIQUE Attempts to transplant the jejunum in two stages should be avoided because the raw edge of the mesentery of the mobilized loop becomes so intimately adherent to the surrounding tissues that subsequent freeing to finish the procedure at a second stage becomes virtually impossible without injury to the vascular arches.

FIGURE 288 Diagram showing the first stage of a multiple stage operation using a combination of skin tube and jejunal transplant to the subcutaneous tissues. Note loop and mesenteric pedicle brought up through the transverse mesocolon, end-to-side jejunogastric anastomosis, catheter gastrostomy for feeding until the completion of the procedure.



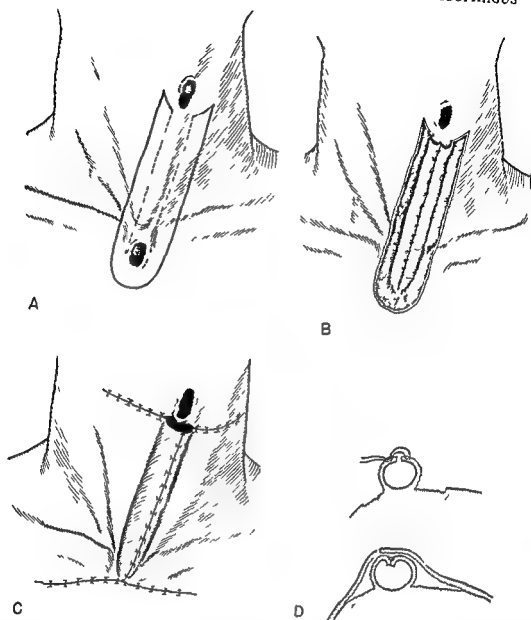


FIGURE 289 Diagram showing the *second stage* of multiple stage operation using skin and jejunal transplant *A* Outline of incision for the construction of skin tube *B*, inner edges turned toward each other and sutured with eversion of edges into the lumen knots of sutures tied on the inside *C* outer skin edges approximated over the skin tube after mobilizing a rectangular flap on each side to permit sliding *D* cross section appearance of *B* and *C*

A two-stage procedure involving the transplantation of a jejunal segment part way up the thorax in a subcutaneous tunnel, supplemented by the interposition of a tube of skin between the proximal end of the esophagus and the end of the jejunal loop, is possible. This method is subject to all of the objections to the use of skin tube replacement as outlined previously. The skin plastic portion of the procedure must usually be accomplished in stages, which prolongs the period of disability and lengthens the hospital stay of the patient. There is also the possibility of stricture formation where the lower end of the skin tube is joined with the jejunum.

There are occasions however, when this modification of the technique may become necessary. No detailed description of the technique need be given. The mobilization of the jejunum and the performance of the jejuno gastric and jejunojejunal anastomoses are the same as when the one-stage technique described above is used. The construction of the skin tube and its anastomosis to the jejunum and the esophagus are accomplished according to established principles. The technique can be followed in the series of drawings provided in Figures 288, 289, and 290.

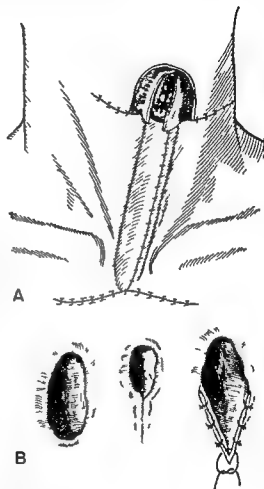


FIGURE 290 : Diagram showing the final stage of multiple stage procedure using skin tube and jejunal transplant. *A* Method of closing the upper end. *B* method of enlarging the stoma if too small because of cicatricial contraction.

Employment of the Colon for Esophageal Replacement

Although a segment of the transverse colon may be used, it is preferable to rely upon the right half of that organ, especially when the anastomosis is to be made in the neck. Utilization of the right colon has certain advantages over the use of a jejunal segment. It is easily reached and technically it is much more readily mobilized than the jejunum. It is, however, subject to failure of its blood supply, and the abnormalities of the vascular pattern may make it necessary to abandon the procedure. For the same reason also, it is not wise to use the colon for anastomoses performed within the mediastinum. A leak at the suture line in this location is almost certain to end fatally or at least to provide

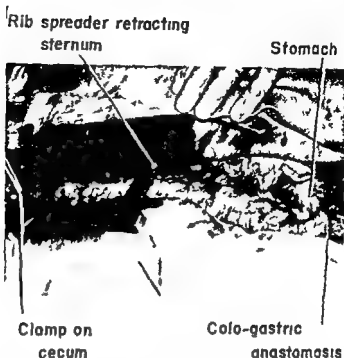


FIGURE 291 Photograph showing the exposure of the anterior mediastinum for bringing up a colon transplant (operation)

almost insurmountable difficulties if the patient survives. Leakages in the neck, however, usually heal without incident.

Preliminary antibiotic preparation of the bowel is highly desirable. If the patient has a gastrostomy, this can be accomplished easily using Sulfathalidine in solution. Neomycin may be used as a substitute, but the results are rarely as satisfactory.

The same type of incision is employed as is used for a jejunal transplant. The entire right colon is freed by severing the lateral peritoneal attachment. If the appendix is present, it should be removed. The terminal ileum is divided and the distal end is closed. This part of the procedure, however, must be accomplished in such a manner as to maintain the continuity of any vascular anastomoses between the ileum and the cecum. If this step is not observed, there is danger of focal necrosis of the cecum in some patients. In this event, the cecum must be resected to a point where the blood supply is adequate. This necessity may spoil the entire procedure by causing too much shortening of the bowel.

The ileocolic and right colic arteries and veins are divided and tied close to their origin so as to preserve the continuity of their anastomotic vascular channels.

The right colon is then swung up with the cecum uppermost and drawn out of the incision over the anterior chest wall to test its length, which is usually adequate. In order to avoid obstructing the antral portion of the stomach by the pressure of the vascular pedicle of the isolated bowel, the loop is brought up behind the stomach and forward through an opening made in the gastrocolic

ligament This leaves the vital middle colic artery behind the antrum instead of in front

The anterior mediastinal space is prepared either by blunt dissection from above and below or preferably by enlarging the incision upward through the sternum (Fig. 291) These techniques are the same as was described above for the employment of jejunum (page 407)

In the same manner, also the cervical esophagus is divided, its distal end inverted, and the proximal end used for the anastomosis For this purpose one of the longitudinal muscle bands in the cecal wall is chosen after once again making certain by the color of the bowel that its blood supply is adequate Two layers of interrupted fine silk are employed

Attention is then directed toward the performance of the cologastric anastomosis The gastrotomy if present must be closed as described above A suitable spot is chosen on the transverse colon and the bowel is divided, taking great pains to avoid interrupting any but the smallest peripheral vessels The omentum is freed to the point of division The lower end of the isolated segment of colon is anastomosed to the anterior wall of the stomach with two layers of interrupted sutures Although it is desirable to place this anastomosis as close to the fundus of the stomach as possible, this can rarely be accomplished without producing too much traction on the middle colic artery The anastomosis is therefore usually placed on the anterior surface of the antrum near the body of the stomach

As a final step the end of the ileum is anastomosed with the distal end of the transverse colon also with two layers of silk sutures The edge of the ileal mesentery and transverse mesocolon are approximated with a continuous suture of catgut Figure 292 shows a diagram of the procedure

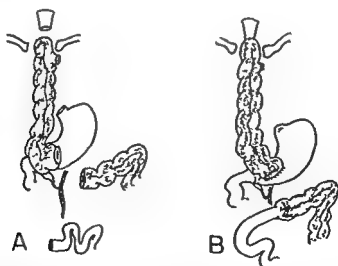


FIGURE 292 Diagram showing the principles involved in the use of the right half of the colon for esophageal substitution. A Preservation of the middle colic artery as a pedicle upon which the colon is turned cecum uppermost for esophagocecal anastomosis The colon is thereby transplanted in an isoperistaltic relation to the alimentary canal B Esophagocecal cologastric and ileocecal anastomoses completed Note The colon is brought up behind the stomach to avoid impingement of the vascular pedicle on the gastric antrum

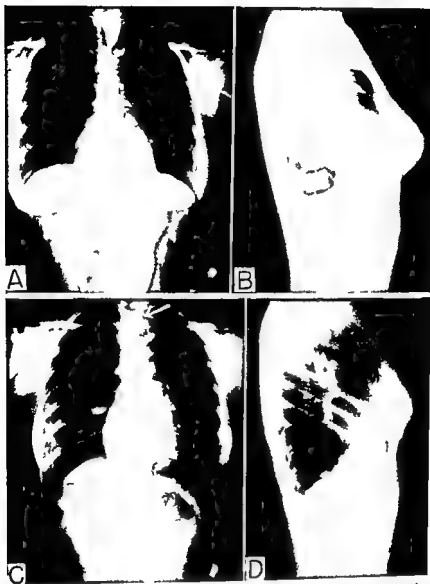


FIGURE 293 Roentgen films of a girl 17 years of age who swallowed lye solution with suicidal intent. *A B* Preoperative esophagograms showing the tremendous extent of the injury. *C and D* frontal and lateral views of barium in the colon segment transplanted to the anterior mediastinum for esophageal substitution. Anastomosis high in the neck (arrow)

Incisional closure is made according to the usual technique. The mediastinum is not drained.

Aftercare The postoperative management follows the same pattern as when the jejunum has been used.

Results Both from the physiological and the mechanical points of view the results of using the right colon for esophageal substitution are excellent. A rapid gain in the patient's weight is to be expected. Discomfort is minimal and digestive complaints are almost nonexistent. One patient experiences a peculiar fluttering sensation in the chest during defecation, caused no doubt by sympathetic reflex peristalsis of the transplanted loop. On roentgen examination no delay in the passage of barium is seen. In fact, the transit is likely to be rather rapid though not unpleasant for the patient.

FIGURE 294 Roentgenogram of barium passing through the transplanted right colon used to substitute for the esophagus in a patient with an unresectable growth in the superior mediastinal segment. Note esophagocecal anastomosis at 1 and cologastric anastomosis at 2 [Wire sutures used to close the sternum at 3]



Figure 293 shows the use of the right colon to supplant the esophagus in an adolescent girl who had a chemical burn of the entire esophagus below the cricoid region. Figure 294 shows a similar case in which the right colon was used to bypass a high thoracic carcinoma of the esophagus which could not be removed.

EMPLOYMENT OF A TUBE PROSTHESIS

As a means of replacing short segments of the esophagus removed by local excision, particularly in certain cases of carcinoma, the substitution of an inert plastic tube has been tried. This technique, however, cannot be expected to provide a large percentage of successes because it violates certain principles of healing which are invoked. In the first place, it goes without saying that the esophagus cannot heal to the inert material. The success of the method, therefore, depends upon maintaining a leak proof union between the divided ends of the esophagus and the tube until enough time has elapsed to permit the periesophageal tissues to build up a barrier of connective tissue which will prevent leakage of the esophageal contents. The possibility of this taking place is diminished by the factors inherent in the method. The esophagus in spite of all efforts to keep it clean is inevitably contaminated with mouth organisms

If necrosis of the ends of the organ where they are attached to the prosthesis should develop, the pathogenic bacteria contained within the lumen may start an infection which leads to mediastinitis and leakage from the union. In the hands of many surgeons, therefore, the employment of a plastic tube has been followed by the occurrence of mediastinal abscesses, empyema of the pleura, esophagocutaneous fistulae, and death. The method cannot be recommended.

CHAPTER 21

Acquired Communications with the Air Passages (Esophagotracheal and Esophagobronchial Fistulae)

THE CLOSE physical proximity of the esophagus to the trachea and major bronchi explains the frequency of occurrence of an abnormal communication between it and the respiratory tract. Usually this condition is a late complication, the prognostic significance of which is serious. This is especially so because the underlying cause is almost always ominous in itself. Occasional instances of healing have, however, been recorded.

Tracheo-esophageal fistulae of congenital origin are described elsewhere (Chapter 6).

The term, *esophagorespiratory communication*, is self explanatory. Although certain authors use the term *perforation* or *fistula* for all cases, the latter denomination has a precise anatomical significance and should actually be reserved for the cases in which a true fistulous tract exists. In other instances the loss of substance between the esophagus and the trachea, for example, is so vast that it is a real abuse of the language to speak of it as a fistula.

The etiological aspects are extremely varied and the similarities between cases depend much more upon their clinical characteristics than upon the lesions which are their immediate cause. The occurrence is a complication of any one of a number of esophageal, tracheal, bronchial, or mediastinal disorders. For this reason it is convenient here to present only the essential characteristics in order to avoid a repetition of the material presented in the special chapters devoted to each of the conditions mentioned.

Etiology

The usual considerations as to the age and sex of the patient are of no importance since the condition under consideration is merely a compilation of affections of varying types, each with its own etiological characteristics.

The cause of communications between the esophagus and the trachea or bronchi may be on any of three bases, depending upon the location of the initial lesion.

1 *Lesions Arising in the Wall of the Esophagus* These may ulcerate the wall of the esophagus during the course of their development and enter the respiratory tract by way of a more or less elongated tract. This is by far the most frequent situation, accounting for over 80 per cent of the cases.

2 *Lesions of Mediastinal Origin* If the initial lesion has arisen in any of the periesophageal, peritracheal, or peribronchial groups of mediastinal lymph nodes or even in the connective tissues, the communication usually develops in two stages. The disease process, usually a collection of pus, opens at first into one of the passages, then secondarily, as it develops, it perforates the wall of the other.

3 *Lesions Arising in the Trachea or Bronchus* Affections of this major portion of the respiratory tree which may be likely to cause perforations are rare. In fact, there are hardly any except new growths. For this reason this category is the least important numerically, although its clinical interest is great.

Lesions of the Esophagus

It is necessary to review for this purpose all the pathology of the esophagus, but actually the usual etiology of perforations can be summarized under several essential headings.

INJURIES *Accidental occurrences* of various sorts may create a communication between organs which lie as close together as the esophagus and the trachea in the superior mediastinum and, above all, in their course through the neck. Simultaneous perforations of both organs by a cutting blade or by a pointed object like a piece of glass or a stylet have been observed. Likewise, in time of war, wounds caused by projectiles such as balls or shrapnel are not unusual. In the neck, however, the proximity of the large arterial and venous trunks and in the mediastinum that of essential organs such as the heart and the aorta make death almost immediate or at least very rapid with the symptoms of hemorrhage and asphyxia. Survival long enough to permit clinical observation is exceptional. The discovery of the exact nature of the injury is established at autopsy and always acquires a medicolegal importance.

Operative and therapeutic accidents may give rise to esophagorespiratory communications. During the course of an operation, particularly for carcinoma of the thyroid or for the treatment of some disease of the esophagus itself, perforations may be produced. The same may be true during an esophagoscopy or as a result of an attempt to dilate a stricture. Efforts to extract a foreign body endoscopically sometimes are complicated by a perforation of the esophageal wall. Under any of these circumstances, a complicating penetration into the trachea or a major bronchus is unusual. The false passage is much more often

confined to the mediastinal or cervical connective tissues or extends into the pleural cavity

Injury caused by impacted solid foreign bodies deserves special consideration. It is rare for an esophageal foreign body to possess points or edges of a degree of sharpness sufficient to perforate rapidly the double walled septum between the esophagus and the trachea. More often it is unrecognized old foreign bodies which have perforated the esophageal wall and around which develops a process of encystment that excludes them from the esophageal lumen. Later on, the wall of the air passage in turn becomes infiltrated and eroded because of pressure, thus finally establishing an intercommunication. Such cases are becoming more and more rare due to the perfection of diagnostic procedures and the more widespread employment of endoscopy. Likewise the occasional ulceration into the esophagus of the end of a tracheostomy tube left in place for too long a period of time has become no more than a matter of historical importance.

Erosion by ingested caustic liquids rarely causes injuries of sufficient depth to ulcerate into the respiratory passages. Such a perforation however, can develop at either of two stages of the injury. (1) In the secondary phase when the mucosal eschar is eliminated, a sinus may become established which ultimately breaks into the trachea. (2) In the cicatricial stage after a stenosis has developed, a perforation may result from esophagitis induced by efforts to dilate the stricture.

Perforation caused by a neoplasm which has developed in a chemically induced stricture may be observed as a rare occurrence.

INFECTIONS Syphilis causes a small number of esophagotracheal or bronchial fistulae, but tertiary involvement of the esophagus is unusual. The literature contains reports of two such cases with eventual cure resulting from adequate treatment.

Tuberculosis arising in the esophagus is not mentioned as an authentic cause of the syndrome under consideration. Fistulae doubtless exist but even the most careful anatomical examination does not always make it possible to establish the tracheal or esophageal origin of the initial tuberculous ulcer. The usual origin is the simultaneous erosion into each organ of abscesses resulting from mediastinal lymphadenitis.

Other infections such as actinomycosis and sporotrichosis have been incriminated as exceptional causes.

Fistulae into the respiratory tract resulting from the erosion of nonspecific ulcerations (peptic or otherwise) have not been observed. Chevalier Jackson reported a series of eighty-eight cases of ulcer of this type with no such occurrences.

Exceptionally, erosion through the wall may occur as a result of infection and necrosis in mega-esophagus and esophageal diverticulum.

TUMORS Malignant tumors of esophageal origin are a common cause of fistula formation whereas with benign neoplasms no such occurrence is known. Sarcomas are so rare that from the practical point of view it is carcinoma only which is encountered. In fact, epidermoid carcinoma of the esophagus accounts for two thirds or more of the reported fistulae between the esophagus and a bronchus or the trachea. Actually it should be pointed out that this proportion

is doubtless smaller than is the case, because fistulae resulting from carcinoma are so common in occurrence that they are not always reported, whereas those due to other causes are usually the subject of much interest

Fistulization is a frequent complication of cancer of the esophagus, occurring possibly in 30 to 35 per cent of the cases when the growth lies in the segment adjacent to the respiratory passages. It occurs relatively late in the course of the disease, often eight months or more after the onset of symptoms

Lesions Arising in the Mediastinum or Base of the Neck

The relative mobility of the two passageways, both respiratory and alimentary, in their course through the neck makes it possible for them to avoid to a considerable extent the dangerous proximity of a collection of pus or of a tumor, either of which carries with it the possibility of developing simultaneous perforations. Carcinoma arising in the thyroid, however, may cause such a fistula

In the mediastinum, where the two organs are closely attached to each other, the situation is different. In the superior mediastinum, cold abscesses from Pott's disease or other suppurative collections may lead to fistula formation. In the midportion of the mediastinum each organ, depending upon the nature of the pathological condition present, is susceptible to the development of esophagorespiratory communications. In fact, this is the location of the largest number of fistulae into the trachea or bronchi.

To understand the reasons for this, one should recall the topographical anatomy of the subcarinal region. Above are the trachea and the origins of the major bronchi. Below are the primary branches of the pulmonary artery, and in the posterior portion of this irregularly lozenge-shaped area lies the esophagus. Within the bounds of this area is a large group of lymph nodes. Other nodes lie between the esophagus and the trachea or bronchi (paratracheal and peribronchial groups). Finally, the arch of the aorta circles over the left main bronchus and the esophagus. The left main bronchus is in more immediate contact with the esophagus than the right which, except in mega esophagus, rarely has any direct contact with this organ.

Occasionally an aortic aneurysm may give rise to a broncho-esophageal communication. There is little clinical history in such cases. The erosion produced by the aneurysmal sac enters the two passageways simultaneously and death results from a cataclysmic hemorrhage.

More important is *suppurative mediastinal lymphadenitis*. This includes the respiratory passage and the esophagus in an inflammatory mass which finally erodes first into one and then into the other of these organs. Tuberculosis is the usual cause of such a lesion. Much less often are *splenic lymphadenopathy* and metastatic carcinoma secondary to pulmonary or esophageal primary lesions. Metastases from other sites may on rare occasions be at fault.

In rare instances an abscess caused by the usual pyogenic organisms may be a source of fistulization. Few well substantiated cases of this are known.

Finally, mediastinitis from various causes such as necrotic primary tumors of the mediastinum, carcinoma or abscess of the lung or occasionally empyema may under exceptional circumstances be the cause. In any such case,

however, the diagnosis, though it may be suspected during the life of the patient, must be proved by the anatomical and bacteriological findings at autopsy

Lesions Arising in the Trachea or Bronchi

This group of cases includes primary neoplasms of the trachea or bronchi, foreign bodies lodged in the respiratory tract, syphilis, and tuberculosis

Carcinoma of the trachea, located most often in the upper or lower thirds of the organ, tends to bulge into the lumen rather than to penetrate deeply through the wall of the tract. Sometimes, however, it may invade the wall of the esophagus and as soon as this occurs a fistula becomes almost inevitable. Baratoux gathered observations on fifty-six cases of carcinoma arising in the trachea, in only one of which he found a fistula into the esophagus. The importance of this possibility is, however, limited because of the rarity of this disease.

Carcinomata of the major bronchi resemble those of the trachea in their behavior, but as they develop in a passageway the caliber of which is somewhat smaller, their symptoms are noticed earlier and are more acute. Furthermore the development of such a lesion usually takes place along the bronchopulmonary tree rather than in the direction of the esophagus, fistulization from these tumors is a rare occurrence.

So far as *foreign bodies* in the respiratory tract are concerned those with sharp points which may become fixed in the wall of the trachea may at the same time penetrate both the tracheal and esophageal walls. In such cases, however, there is rarely a functional intercommunication.

Syphilis of the trachea and bronchi is not as unusual as one might suppose but although a gumma may erode into both the esophagus and the respiratory tract to form a fistula this eventuality is exceptional. A few observations only are available for reference, notably those of Bazy and of Chene concerning the trachea and of Peco concerning the right main bronchus.

The ulcerating form of *tracheal tuberculosis* is the only type likely to cause perforations. It is encountered usually in cachectic patients whose resistance is low and who have cavities. Furthermore, the condition is likely to be overlooked because it almost always occurs in the presence of extensive laryngeal involvement which masks the symptoms. It is therefore almost always merely an autopsy finding.

Tuberculous bronchial ulceration as a cause of fistula into the esophagus has not been reported.

Certain anomalies which might give rise to tracheo-esophageal fistula, other than those discussed in Chapter 6, should be mentioned here because their congenital origin is doubtful. These consist of rounded perforations between the esophagus and the tracheobronchial tree. They may be the result of the breakdown into each organ of a small tracheal or bronchial cyst of possible congenital origin. Little is known about them.

There is also a small number of fistulae the etiology of which is not understood, which are usually narrow and more important morphologically than clinically. They present no symptoms even over a long period of years or even a lifetime (E. Negus). One is tempted without actual proof to attribute to them an embryological significance.

is doubtless smaller than is the case, because fistulae resulting from carcinoma are so common in occurrence that they are not always reported, whereas those due to other causes are usually the subject of much interest

Fistulization is a frequent complication of cancer of the esophagus, occurring possibly in 30 to 35 per cent of the cases when the growth lies in the segment adjacent to the respiratory passages. It occurs relatively late in the course of the disease, often eight months or more after the onset of symptoms

Lesions Arising in the Mediastinum or Base of the Neck

The relative mobility of the two passageways, both respiratory and alimentary, in their course through the neck makes it possible for them to avoid to a considerable extent the dangerous proximity of a collection of pus or of a tumor, either of which carries with it the possibility of developing simultaneous perforations. Carcinoma arising in the thyroid, however, may cause such a fistula

In the mediastinum, where the two organs are closely attached to each other, the situation is different. In the superior mediastinum, cold abscesses from Pott's disease or other suppurative collections may lead to fistula formation. In the midportion of the mediastinum each organ, depending upon the nature of the pathological condition present, is susceptible to the development of esophagorespiratory communications. In fact, this is the location of the largest number of fistulae into the trachea or bronchi.

To understand the reasons for this, one should recall the topographical anatomy of the subcarinal region. Above are the trachea and the origins of the major bronchi. Below are the primary branches of the pulmonary artery, and in the posterior portion of this irregularly lozenge-shaped area lies the esophagus. Within the bounds of this area is a large group of lymph nodes. Other nodes lie between the esophagus and the trachea or bronchi (paratracheal and peribronchial groups). Finally, the arch of the aorta circles over the left main bronchus and the esophagus. The left main bronchus is in more immediate contact with the esophagus than the right which, except in mega esophagus, rarely has any direct contact with this organ.

Occasionally an aortic aneurysm may give rise to a broncho esophageal communication. There is little clinical history in such cases. The erosion produced by the aneurysmal sac enters the two passageways simultaneously and death results from a cataclysmic hemorrhage.

More important is *suppurative mediastinal lymphadenitis*. This includes the respiratory passage and the esophagus in an inflammatory mass which finally erodes first into one and then into the other of these organs. *Tuberculosis* is the usual cause of such a lesion. Much less often are *syphilitic lymphadenopathy* and metastatic carcinoma secondary to pulmonary or esophageal primary lesions. Metastases from other sites may on rare occasions be at fault.

In rare instances an abscess caused by the usual pyogenic organisms may be a source of fistulization. Few well substantiated cases of this are known.

Finally, *mediastinitis* from various causes such as necrotic primary tumors of the mediastinum, carcinoma or abscess of the lung or occasionally empyema may under exceptional circumstances be the cause. In any such case,

however, the diagnosis, though it may be suspected during the life of the patient, must be proved by the anatomical and bacteriological findings at autopsy

Lesions Arising in the Trachea or Bronchi

This group of cases includes primary neoplasms of the trachea or bronchi, foreign bodies lodged in the respiratory tract syphilis and tuberculosis

Carcinoma of the trachea, located most often in the upper or lower thirds of the organ, tends to bulge into the lumen rather than to penetrate deeply through the wall of the tract Sometimes, however, it may invade the wall of the esophagus and as soon as this occurs a fistula becomes almost inevitable Baratoux gathered observations on fifty six cases of carcinoma arising in the trachea, in only one of which he found a fistula into the esophagus The importance of this possibility is however, limited because of the rarity of this disease

Carcinomata of the major bronchi resemble those of the trachea in their behavior but as they develop in a passageway the caliber of which is somewhat smaller, their symptoms are noticed earlier and are more acute Furthermore, the development of such a lesion usually takes place along the bronchopulmonary tree rather than in the direction of the esophagus, fistulization from these tumors is a rare occurrence

So far as *foreign bodies* in the respiratory tract are concerned, those with sharp points which may become fixed in the wall of the trachea may at the same time penetrate both the tracheal and esophageal walls In such cases, however there is rarely a functional intercommunication

Syphilis of the trachea and bronchi is not as unusual as one might suppose but although a gumma may erode into both the esophagus and the respiratory tract to form a fistula, this eventuality is exceptional A few observations only are available for reference notably those of Bazy and of Chene concerning the trachea and of Peco concerning the right main bronchus

The ulcerating form of *tracheal tuberculosis* is the only type likely to cause perforations It is encountered usually in cachectic patients whose resistance is low and who have cavities Furthermore, the condition is likely to be overlooked because it almost always occurs in the presence of extensive laryngeal involvement which masks the symptoms It is therefore almost always merely an autopsy finding

Tuberculous bronchial ulceration as a cause of fistula into the esophagus has not been reported

Certain anomalies which might give rise to tracheo-esophageal fistula other than those discussed in Chapter 6 should be mentioned here because their congenital origin is doubtful These consist of rounded perforations between the esophagus and the tracheobronchial tree They may be the result of the breakdown into each organ of a small tracheal or bronchial cyst of possible congenital origin Little is known about them

There is also a small number of fistulae, the etiology of which is not understood, which are usually narrow and more important morphologically than clinically They present no symptoms even over a long period of years or even a lifetime (E. Negus) One is tempted without actual proof to attribute to them an embryological significance

is doubtless smaller than is the case, because fistulae resulting from carcinoma are so common in occurrence that they are not always reported, whereas those due to other causes are usually the subject of much interest

Fistulization is a frequent complication of cancer of the esophagus, occurring possibly in 30 to 35 per cent of the cases when the growth lies in the segment adjacent to the respiratory passages. It occurs relatively late in the course of the disease, often eight months or more after the onset of symptoms

Lesions Arising in the Mediastinum or Base of the Neck

The relative mobility of the two passageways, both respiratory and alimentary, in their course through the neck makes it possible for them to avoid to a considerable extent the dangerous proximity of a collection of pus or of a tumor, either of which carries with it the possibility of developing simultaneous perforations. Carcinoma arising in the thyroid, however, may cause such a fistula

In the mediastinum, where the two organs are closely attached to each other, the situation is different. In the superior mediastinum, cold abscesses from Pott's disease or other suppurative collections may lead to fistula formation. In the midportion of the mediastinum each organ, depending upon the nature of the pathological condition present, is susceptible to the development of esophagorespiratory communications. In fact, this is the location of the largest number of fistulae into the trachea or bronchi

To understand the reasons for this, one should recall the topographical anatomy of the subcarinal region. Above are the trachea and the origins of the major bronchi. Below are the primary branches of the pulmonary artery, and in the posterior portion of this irregularly lozenge-shaped area lies the esophagus. Within the bounds of this area is a large group of lymph nodes. Other nodes lie between the esophagus and the trachea or bronchi (paratracheal and peribronchial groups). Finally, the arch of the aorta circles over the left main bronchus and the esophagus. The left main bronchus is in more immediate contact with the esophagus than the right which, except in mega esophagus, rarely has any direct contact with this organ

Occasionally an aortic aneurysm may give rise to a broncho-esophageal communication. There is little clinical history in such cases. The erosion produced by the aneurysmal sac enters the two passageways simultaneously and death results from a cataclysmic hemorrhage

More important is suppurative mediastinal lymphadenitis. This includes the respiratory passage and the esophagus in an inflammatory mass which finally erodes first into one and then into the other of these organs. Tuberculosis is the usual cause of such a lesion. Much less often are syphilitic lymphadenopathy and metastatic carcinoma secondary to pulmonary or esophageal primary lesions. Metastases from other sites may on rare occasions be at fault

In rare instances an abscess caused by the usual pyogenic organisms may be a source of fistulization. Few well substantiated cases of this are known

Finally, mediastinitis from various causes such as necrotic primary tumors of the mediastinum, carcinoma or abscess of the lung or occasionally empyema may under exceptional circumstances be the cause. In any such case,

however, the diagnosis, though it may be suspected during the life of the patient must be proved by the anatomical and bacteriological findings at autopsy

Lesions Arising in the Trachea or Bronchi

This group of cases includes primary neoplasms of the trachea or bronchi, foreign bodies lodged in the respiratory tract, syphilis and tuberculosis

Carcinoma of the trachea, located most often in the upper or lower thirds of the organ tends to bulge into the lumen rather than to penetrate deeply through the wall of the tract. Sometimes, however, it may invade the wall of the esophagus and as soon as this occurs a fistula becomes almost inevitable. Baratoux gathered observations on fifty-six cases of carcinoma arising in the trachea, in only one of which he found a fistula into the esophagus. The importance of this possibility is however, limited because of the rarity of this disease.

Carcinomata of the major bronchi resemble those of the trachea in their behavior, but as they develop in a passageway the caliber of which is somewhat smaller their symptoms are noticed earlier and are more acute. Furthermore the development of such a lesion usually takes place along the bronchopulmonary tree rather than in the direction of the esophagus, fistulization from these tumors is a rare occurrence.

So far as *foreign bodies* in the respiratory tract are concerned, those with sharp points which may become fixed in the wall of the trachea may at the same time penetrate both the tracheal and esophageal walls. In such cases, however, there is rarely a functional intercommunication.

Syphilis of the trachea and bronchi is not as unusual as one might suppose but although a gumma may erode into both the esophagus and the respiratory tract to form a fistula, this eventuality is exceptional. A few observations only are available for reference notably those of Bazy and of Chene concerning the trachea and of Peco concerning the right main bronchus.

The ulcerating form of *tracheal tuberculosis* is the only type likely to cause perforations. It is encountered usually in cachectic patients whose resistance is low and who have cavities. Furthermore, the condition is likely to be overlooked because it almost always occurs in the presence of extensive laryngeal involvement which masks the symptoms. It is therefore almost always merely an autopsy finding.

Tuberculous bronchial ulceration as a cause of fistula into the esophagus has not been reported.

Certain anomalies which might give rise to tracheo-esophageal fistula other than those discussed in Chapter 6 should be mentioned here because their congenital origin is doubtful. These consist of rounded perforations between the esophagus and the tracheobronchial tree. They may be the result of the breakdown into each organ of a small tracheal or bronchial cyst of possible congenital origin. Little is known about them.

There is also a small number of fistulae, the etiology of which is not understood, which are usually narrow and more important morphologically than clinically. They present no symptoms even over a long period of years or even a lifetime (E. Negus). One is tempted without actual proof to attribute to them an embryological significance.

is doubtless smaller than is the case, because fistulae resulting from carcinoma are so common in occurrence that they are not always reported, whereas those due to other causes are usually the subject of much interest

Fistulization is a frequent complication of cancer of the esophagus, occurring possibly in 30 to 35 per cent of the cases when the growth lies in the segment adjacent to the respiratory passages. It occurs relatively late in the course of the disease, often eight months or more after the onset of symptoms

Lesions Arising in the Mediastinum or Base of the Neck

The relative mobility of the two passageways, both respiratory and alimentary, in their course through the neck makes it possible for them to avoid to a considerable extent the dangerous proximity of a collection of pus or of a tumor, either of which carries with it the possibility of developing simultaneous perforations. Carcinoma arising in the thyroid, however, may cause such a fistula

In the mediastinum, where the two organs are closely attached to each other, the situation is different. In the superior mediastinum, cold abscesses from Pott's disease or other suppurative collections may lead to fistula formation. In the midportion of the mediastinum each organ, depending upon the nature of the pathological condition present, is susceptible to the development of esophagorespiratory communications. In fact, this is the location of the largest number of fistulae into the trachea or bronchi.

To understand the reasons for this, one should recall the topographical anatomy of the subcarinal region. Above are the trachea and the origins of the major bronchi. Below are the primary branches of the pulmonary artery, and in the posterior portion of this irregularly lozenge-shaped area lies the esophagus. Within the bounds of this area is a large group of lymph nodes. Other nodes lie between the esophagus and the trachea or bronchi (paratracheal and peribronchial groups). Finally, the arch of the aorta circles over the left main bronchus and the esophagus. The left main bronchus is in more immediate contact with the esophagus than the right which, except in mega esophagus, rarely has any direct contact with this organ.

Occasionally an *aortic aneurysm* may give rise to a broncho esophageal communication. There is little clinical history in such cases. The erosion produced by the aneurysmal sac enters the two passageways simultaneously and death results from a cataclysmic hemorrhage.

More important is *suppurative mediastinal lymphadenitis*. This includes the respiratory passage and the esophagus in an inflammatory mass which finally erodes first into one and then into the other of these organs. Tuberculosis is the usual cause of such a lesion. Much less often are *siphilic lymphadenopathy* and metastatic carcinoma secondary to pulmonary or esophageal primary lesions. Metastases from other sites may on rare occasions be at fault.

In rare instances an abscess caused by the usual pyogenic organisms may be a source of fistulization. Few well substantiated cases of this are known.

Finally, mediastinitis from various causes such as necrotic primary tumors of the mediastinum, carcinoma or abscess of the lung or occasionally empyema may under exceptional circumstances be the cause. In any such case,

however, the diagnosis, though it may be suspected during the life of the patient must be proved by the anatomical and bacteriological findings at autopsy

Lesions Arising in the Trachea or Bronchi

This group of cases includes primary neoplasms of the trachea or bronchi, foreign bodies lodged in the respiratory tract syphilis and tuberculosis

Carcinoma of the trachea, located most often in the upper or lower thirds of the organ tends to bulge into the lumen rather than to penetrate deeply through the wall of the tract Sometimes, however, it may invade the wall of the esophagus and as soon as this occurs a fistula becomes almost inevitable Baratoux gathered observations on fifty-six cases of carcinoma arising in the trachea, in only one of which he found a fistula into the esophagus The importance of this possibility is however, limited because of the rarity of this disease

Carcinomata of the major bronchi resemble those of the trachea in their behavior but as they develop in a passageway the caliber of which is somewhat smaller their symptoms are noticed earlier and are more acute Furthermore, the development of such a lesion usually takes place along the bronchopulmonary tree rather than in the direction of the esophagus, fistulization from these tumors is a rare occurrence

So far as *foreign bodies* in the respiratory tract are concerned, those with sharp points which may become fixed in the wall of the trachea may at the same time penetrate both the tracheal and esophageal walls In such cases, however there is rarely a functional intercommunication

Syphilis of the trachea and bronchi is not as unusual as one might suppose but although a gumma may erode into both the esophagus and the respiratory tract to form a fistula, this eventuality is exceptional A few observations only are available for reference notably those of Bazzy and of Chene concerning the trachea and of Peco concerning the right main bronchus

The ulcerating form of *tracheal tuberculosis* is the only type likely to cause perforations It is encountered usually in cachectic patients whose resistance is low and who have cavities Furthermore, the condition is likely to be overlooked because it almost always occurs in the presence of extensive laryngeal involvement which masks the symptoms It is therefore almost always merely an autopsy finding

Tuberculous bronchial ulceration as a cause of fistula into the esophagus has not been reported

Certain anomalies which might give rise to tracheo-esophageal fistula other than those discussed in Chapter 6 should be mentioned here because their congenital origin is doubtful These consist of rounded perforations between the esophagus and the tracheobronchial tree They may be the result of the breakdown into each organ of a small tracheal or bronchial cyst of possible congenital origin Little is known about them

There is also a small number of fistulae the etiology of which is not understood, which are usually narrow and more important morphologically than clinically They present no symptoms even over a long period of years or even a lifetime (E. Negus) One is tempted without actual proof to attribute to them an embryological significance

is doubtless smaller than is the case, because fistulae resulting from carcinoma are so common in occurrence that they are not always reported, whereas those due to other causes are usually the subject of much interest

Fistulization is a frequent complication of cancer of the esophagus, occurring possibly in 30 to 35 per cent of the cases when the growth lies in the segment adjacent to the respiratory passages. It occurs relatively late in the course of the disease, often eight months or more after the onset of symptoms

Lesions Arising in the Mediastinum or Base of the Neck

The relative mobility of the two passageways, both respiratory and alimentary, in their course through the neck makes it possible for them to avoid to a considerable extent the dangerous proximity of a collection of pus or of a tumor, either of which carries with it the possibility of developing simultaneous perforations. Carcinoma arising in the thyroid, however, may cause such a fistula

In the mediastinum, where the two organs are closely attached to each other, the situation is different. In the superior mediastinum, cold abscesses from Pott's disease or other suppurative collections may lead to fistula formation. In the midportion of the mediastinum each organ, depending upon the nature of the pathological condition present, is susceptible to the development of esophagorespiratory communications. In fact, this is the location of the largest number of fistulae into the trachea or bronchi.

To understand the reasons for this, one should recall the topographical anatomy of the subcarinal region. Above are the trachea and the origins of the major bronchi. Below are the primary branches of the pulmonary artery, and in the posterior portion of this irregularly lozenge-shaped area lies the esophagus. Within the bounds of this area is a large group of lymph nodes. Other nodes lie between the esophagus and the trachea or bronchi (paratracheal and peribronchial groups). Finally, the arch of the aorta circles over the left main bronchus and the esophagus. The left main bronchus is in more immediate contact with the esophagus than the right which, except in mega esophagus, rarely has any direct contact with this organ.

Occasionally an aortic aneurysm may give rise to a broncho esophageal communication. There is little clinical history in such cases. The erosion produced by the aneurysmal sac enters the two passageways simultaneously and death results from a cataclysmic hemorrhage.

More important is *suppurative mediastinal lymphadenitis*. This includes the respiratory passage and the esophagus in an inflammatory mass which finally erodes first into one and then into the other of these organs. *Tuberculosis* is the usual cause of such a lesion. Much less often are *siphilotic lymphadenopathy* and metastatic carcinoma secondary to pulmonary or esophageal primary lesions. Metastases from other sites may on rare occasions be at fault.

In rare instances an abscess caused by the usual pyogenic organisms may be a source of fistulization. Few well substantiated cases of this are known.

Finally, mediastinitis from various causes such as necrotic primary tumors of the mediastinum, carcinoma or abscess of the lung or occasionally empyema may under exceptional circumstances be the cause. In any such case

however, the diagnosis, though it may be suspected during the life of the patient must be proved by the anatomical and bacteriological findings at autopsy

Lesions Arising in the Trachea or Bronchi

This group of cases includes primary neoplasms of the trachea or bronchi, foreign bodies lodged in the respiratory tract syphilis and tuberculosis

Carcinoma of the trachea, located most often in the upper or lower thirds of the organ tends to bulge into the lumen rather than to penetrate deeply through the wall of the tract Sometimes, however, it may invade the wall of the esophagus and as soon as this occurs a fistula becomes almost inevitable Baratoux gathered observations on fifty-six cases of carcinoma arising in the trachea, in only one of which he found a fistula into the esophagus The importance of this possibility is however, limited because of the rarity of this disease

Carcinomata of the major bronchi resemble those of the trachea in their behavior, but as they develop in a passageway the caliber of which is somewhat smaller, their symptoms are noticed earlier and are more acute Furthermore the development of such a lesion usually takes place along the bronchopulmonary tree rather than in the direction of the esophagus, fistulization from these tumors is a rare occurrence

So far as *foreign bodies* in the respiratory tract are concerned, those with sharp points which may become fixed in the wall of the trachea may at the same time penetrate both the tracheal and esophageal walls In such cases however, there is rarely a functional intercommunication

Syphilis of the trachea and bronchi is not as unusual as one might suppose but although a gumma may erode into both the esophagus and the respiratory tract to form a fistula, this eventuality is exceptional A few observations only are available for reference notably those of Bazy and of Chene concerning the trachea and of Peco concerning the right main bronchus

The ulcerating form of *tracheal tuberculosis* is the only type likely to cause perforations It is encountered usually in cachectic patients whose resistance is low and who have cavities Furthermore, the condition is likely to be overlooked because it almost always occurs in the presence of extensive laryngeal involvement which masks the symptoms It is therefore almost always merely an autopsy finding

Tuberculous bronchial ulceration as a cause of fistula into the esophagus has not been reported

Certain anomalies which might give rise to tracheo-esophageal fistula other than those discussed in Chapter 6 should be mentioned here because their congenital origin is doubtful These consist of rounded perforations between the esophagus and the tracheobronchial tree They may be the result of the breakdown into each organ of a small tracheal or bronchial cyst of possible congenital origin Little is known about them

There is also a small number of fistulae the etiology of which is not understood, which are usually narrow and more important morphologically than clinically They present no symptoms even over a long period of years or even a lifetime (E. Negus) One is tempted without actual proof to attribute to them an embryological significance

is doubtless smaller than is the case, because fistulae resulting from carcinoma are so common in occurrence that they are not always reported, whereas those due to other causes are usually the subject of much interest

Fistulization is a frequent complication of cancer of the esophagus, occurring possibly in 30 to 35 per cent of the cases when the growth lies in the segment adjacent to the respiratory passages. It occurs relatively late in the course of the disease, often eight months or more after the onset of symptoms

Lesions Arising in the Mediastinum or Base of the Neck

The relative mobility of the two passageways, both respiratory and alimentary, in their course through the neck makes it possible for them to avoid to a considerable extent the dangerous proximity of a collection of pus or of a tumor, either of which carries with it the possibility of developing simultaneous perforations. Carcinoma arising in the thyroid, however, may cause such a fistula

In the mediastinum, where the two organs are closely attached to each other, the situation is different. In the superior mediastinum, cold abscesses from Pott's disease or other suppurative collections may lead to fistula formation. In the midportion of the mediastinum each organ, depending upon the nature of the pathological condition present, is susceptible to the development of esophagorespiratory communications. In fact, this is the location of the largest number of fistulae into the trachea or bronchi.

To understand the reasons for this, one should recall the topographical anatomy of the subcarinal region. Above are the trachea and the origins of the major bronchi. Below are the primary branches of the pulmonary artery, and in the posterior portion of this irregularly lozenge-shaped area lies the esophagus. Within the bounds of this area is a large group of lymph nodes. Other nodes lie between the esophagus and the trachea or bronchi (paratracheal and peribronchial groups). Finally, the arch of the aorta circles over the left main bronchus and the esophagus. The left main bronchus is in more immediate contact with the esophagus than the right which, except in mega-esophagus, rarely has any direct contact with this organ.

Occasionally an aortic aneurysm may give rise to a broncho-esophageal communication. There is little clinical history in such cases. The erosion produced by the aneurysmal sac enters the two passageways simultaneously and death results from a cataclysmic hemorrhage.

More important is *suppurative mediastinal lymphadenitis*. This includes the respiratory passage and the esophagus in an inflammatory mass which finally erodes first into one and then into the other of these organs. *Tuberculosis* is the usual cause of such a lesion. Much less often are *sylphitic lymphadenopathy* and *metastatic carcinoma* secondary to pulmonary or esophageal primary lesions. Metastases from other sites may on rare occasions be at fault.

In rare instances an abscess caused by the usual pyogenic organisms may be a source of fistulization. Few well substantiated cases of this are known.

Finally, *mediastinitis* from various causes such as necrotic primary tumors of the mediastinum, carcinoma or abscess of the lung or occasionally emphysema may under exceptional circumstances be the cause. In any such case,

however, the diagnosis, though it may be suspected during the life of the patient must be proved by the anatomical and bacteriological findings at autopsy

Lesions Arising in the Trachea or Bronchi

This group of cases includes primary neoplasms of the trachea or bronchi, foreign bodies lodged in the respiratory tract, syphilis, and tuberculosis

Carcinoma of the trachea located most often in the upper or lower thirds of the organ, tends to bulge into the lumen rather than to penetrate deeply through the wall of the tract. Sometimes however, it may invade the wall of the esophagus and as soon as this occurs a fistula becomes almost inevitable. Baratoux gathered observations on fifty-six cases of carcinoma arising in the trachea in only one of which he found a fistula into the esophagus. The importance of this possibility is however limited because of the rarity of this disease.

Carcinomata of the major bronchi resemble those of the trachea in their behavior, but as they develop in a passageway the caliber of which is somewhat smaller, their symptoms are noticed earlier and are more acute. Furthermore the development of such a lesion usually takes place along the bronchopulmonary tree rather than in the direction of the esophagus. fistulization from these tumors is a rare occurrence.

So far as *foreign bodies* in the respiratory tract are concerned, those with sharp points which may become fixed in the wall of the trachea may at the same time penetrate both the tracheal and esophageal walls. In such cases however, there is rarely a functional intercommunication.

Syphilis of the trachea and bronchi is not as unusual as one might suppose, but although a gumma may erode into both the esophagus and the respiratory tract to form a fistula this eventuality is exceptional. A few observations only are available for reference, notably those of Bazy and of Chene concerning the trachea and of Peco concerning the right main bronchus.

The ulcerating form of *tracheal tuberculosis* is the only type likely to cause perforations. It is encountered usually in cachectic patients whose resistance is low and who have cavities. Furthermore, the condition is likely to be overlooked because it almost always occurs in the presence of extensive laryngeal involvement which masks the symptoms. It is therefore almost always merely an autopsy finding.

Tuberculous bronchial ulceration as a cause of fistula into the esophagus has not been reported.

Certain anomalies which might give rise to tracheo-esophageal fistula other than those discussed in Chapter 6, should be mentioned here because their congenital origin is doubtful. These consist of rounded perforations between the esophagus and the tracheobronchial tree. They may be the result of the breakdown into each organ of a small tracheal or bronchial cyst of possible congenital origin. Little is known about them.

There is also a small number of fistulae the etiology of which is not understood which are usually narrow and more important morphologically than clinically. They present no symptoms even over a long period of years or even a lifetime (E. Negus). One is tempted without actual proof to attribute to them an embryological significance.

Pathogenesis and Pathological Anatomy

The study of the pathogenesis is bound up with that of the underlying abnormality or disease condition and cannot be separated from it. There is no point, therefore, in presenting here a chapter on the pathology of the condition. The discussion will be confined to certain details regarding the physical characteristics of these fistulae or perforations and the anatomico-pathological conditions peculiar to communications between the esophagus and the tracheobronchial tree.


RELATIVE FREQUENCY Perforations into the trachea or bronchi represent more than three-fourths of the fistulae between the esophagus and the air passages. Tracheal fistulae are twice as common as those involving a bronchus.

LOCATION Perforations into the upper third of the trachea are the least frequent. Those of the middle third are encountered much more often. Finally, for the topographical and etiological reasons mentioned previously, perforations into the inferior third of the trachea, in the carinal region, or in the major bronchi make up approximately one-half of the tracheobronchial esophageal fistulae. For similar reasons the left bronchus is more often involved than the right.

NUMBER Perforations are usually single, but exceptions to this rule are not uncommon. Fistulous communications due to the usual causes (cancer and lymph node suppuration) are quite often multiple. The reason for this is easy to understand. In these cases the communication never (or at least only rarely occurs) by pure and simple disintegration of the esophagorespiratory tract septum. It becomes established after an intervening stage of perivisceral inflammation due to infection which tends to fix and immobilize the two organs. Since in spite of everything the esophagus, the trachea, and the bronchi are continually subject to the movements resulting from their functional activities, a certain amount of tearing results. This occurrence may cause the production of fissures or sinuses of their walls in various points, which is quite independent of the ulcerating propensity of the causal lesion such as carcinoma. These fissures become confluent and ultimately form extremely complex fistulous tracts which are able to bring about the most minute dissection. Frequently a single break in the continuity of the esophagus is responsible for two or more respiratory tract perforations, sometimes one in the trachea and another in one of the bronchi.

In contrast perforations from esophagitis, from infected diverticula or mega esophagus, from tracheal or bronchial carcinomata, and especially those having a congenital origin are rarely multiple.

PHYSICAL CHARACTERISTICS Without entering upon a prolonged discussion, it is necessary to single out certain rather different anatomical types, three of which may be contrasted, namely carcinomatous perforations, lymphadenopathic fistulae, and latent or incipient communications which may possibly be congenital.

1 *Perforations in the Course of Cancer of the Esophagus* Perforations of the respiratory passages by carcinoma of the esophagus are not always due to the same mechanism. Some are  invasive form of the growth

the malignant ulcer itself erodes the wall of the alimentary canal invades the surrounding connective tissues and gives rise to periesophageal infection caused by the intraluminal bacterial flora. This inflammatory reaction brings about fixation of the esophagus and trachea or one of the bronchi to each other. The deep esophageal ulceration, the margins of which consist of neoplastic proliferations, then evolves into a fistulous tract which ends in the respiratory passage. The opening into the air passages, however, is often very small, always irregular in contour, and more or less obstructed by granulations which may be either inflammatory or neoplastic.

It is in this type of case also that large perforations through the adjacent tracheal and esophageal walls may develop as a result of ulceration. The edges of such a communication are ragged and nodular and extend more and more deeply into each of the organs involved, ending in anatomical alterations the importance of which may not be recognized by the clinician until an autopsy is performed (Fig. 295).

Sometimes, as in the stenosing type of carcinoma, the mechanism and appearance of the fistula are slightly different. In these the perforation does not take place at the site of the neoplastic lesion. It occurs in the dilated proximal portion where the wall of the esophagus develops small erosions because of the constant stagnation and infection. The periesophagitis which follows then affects the trachea or bronchus and as a result a fistula may be formed actually above the carcinomatous stenosis often as a complication of attempts to pass a bougie.

As a third mechanism the intercommunication between the carcinomatous esophagus and the trachea may be by the intermediary of suppurative mediastinal lymphadenitis.



FIGURE 295 Photograph of an autopsy specimen showing an esophagotracheal fistula. A The grooved director in the trachea by passing the fistula opening. B The director passed through the fistula opening from the esophagus behind to the trachea in front. (Bocca)

2 *Fistulae Secondary to Lymph Node Suppuration* No matter what its cause may be, lymph node infection leads to a perinodal inflammation which may engulf or surround both the trachea and the esophagus. Two theories may then be invoked to explain the pathogenesis of fistulization between the two organs. In one it is assumed that at the point where the esophagus is fixed by the inflammation, a so-called "traction diverticulum" is formed. An exacerbation of esophagitis appears which, as it increases in intensity, gradually erodes the mucosa, permitting a secondary infection of the mass of inflamed nodes. This infection, in spite of its primary release by ulceration, gradually approaches the tracheal or bronchial wall, ending in a perforation which creates the fistula. This is the mechanism which is usually assumed to take place.

Conversely it is believed by others that the esophagus, because it is more mobile and plastic, adapts itself more readily to the perivisceral inflammation, whereas the rigid trachea or bronchus which is subject to the unceasing movements of respiration is more likely to become eroded and perforated by the infection. This hypothesis assumes that the perforation of the esophagus occurs only secondarily.

Whatever the mechanism of their formation may be, the appearance of the lesions resulting from these lymph node abscesses is exceedingly variable and difficult to define. In general it is necessary at autopsy to remove a large bloc of mediastinal organs and tissues in which are intimately adherent, or actually buried, the esophagus, the bronchi, the trachea, the aorta, and often the pleura and lungs. A detailed dissection can then be made so as to reduce the mass to smaller dimensions. Finally it is usually by opening one of the organs involved that the fistulous communication can be found and its numerous ramifications explored (Fig 295, A, B). These often defy all description in their complexity. Many times also, multiple fistulae between the two organs are found.

3 *Latent or Incipient Fistulae* on the other hand are characterized by the relative lack or even absence of the element of inflammation. These have clear cut rounded or ovoid margins and often look like a fissure. They vary in size. They may be so small as to prevent the escape of food material into the respiratory tract. Sometimes certain anatomical arrangements exist, such as the presence of a valvular fold or an obliquity of the tract with the bronchial opening located at a higher level than that in the esophagus, which may contribute to this apparent lack of free communication between the airways and the esophagus. These fistulae are probably congenital.

Other lesions may show even different appearances, such as those found in tuberculosis of the esophagus, gummas, or tracheobronchial carcinomata, but their characteristics and their frequency do not merit protracted discussion.

ASSOCIATED LESIONS In addition to the causative lesions which characteristically produce certain types of fistula, it is necessary to mention the associated lesions both near and remote which may be encountered. Among the former are involvement of the vagus and recurrent laryngeal nerves, on rare occasions the phrenic nerve, and involvement of the bronchial or pulmonary vessels, or erosion of the aorta.

Among the latter, pulmonary tuberculosis is the most important, whether primary and associated with the peritracheal or peribronchial adenopathy which

caused the fistula, or secondary, as may occur in the debility which goes with advanced cancer of the esophagus. Others are the bronchopulmonary lesions such as pneumonia or abscess which are so often the complications found at autopsy and which are responsible for the death of the patient.

Clinical Characteristics

1 EARLY SIGNS In close to one half the patients the communication manifests itself suddenly by a bout of violent incessant coughing during the course of a meal. The patient strangles and becomes cyanotic. Sometimes he may even lose consciousness. Repeated coughing provokes the expectoration of small solid particles which are recognized as portions of the food which has just been swallowed. If it is a liquid such as wine the color may be identified in the secretions raised. This makes the diagnosis immediately obvious.

Not infrequently, however, no matter how violent the first episode may have been, the cause is misconstrued and the patient assumes that he has swallowed "the wrong way." The repetition of similar incidents is required to draw attention to the true nature of the difficulty.

In slightly less than one-fourth of the patients the perforation takes place insidiously, and the bout of coughing which usually heralds its occurrence is preceded by premonitory signs which are nevertheless not very characteristic. For example a patient known to have a carcinoma of the esophagus may have for several days experienced a slight dry cough while eating. He may have slight hemoptysis or merely expectorate blood-streaked sputum. He may notice retrosternal pain. In brief, he manifests the signs of tracheobronchial irritation until the day when the first bout of cough and suffocation breaks forth.

2 DURING THE STAGE WHEN THE FISTULA IS WELL ESTABLISHED, there is scarcely any change. At each attempt to swallow, violent coughing, choking, and often cyanosis occur, with relief only after the material which has invaded the air passageways is ejected. These attacks are not always of uniform intensity. Thus when the perforation is still small or when the tract is narrow, liquids may seep into the airway more easily than solids and for a time at least the attacks are induced only by drinking liquids and not by swallowing solids. When there is a large communication, however, the solids are responsible for the most severe attacks of suffocation. Conversely, in such a case liquids may be ingested without much inconvenience if taken in small mouthfuls.

These patients fear the arrival of mealtime. They often invent various means to reduce to the minimum the reaction which they dread by adopting any of a great variety of postures in the effort. Sometimes they find it helpful to lie in a semirecumbent position or bent over backward while eating or drinking. Others fill the lungs with air and close the glottis before swallowing. There are almost as many devices as there are patients who need to develop them. One man developed a technique of swallowing cherry stones to occlude the opening before he began to eat.

During the course of the difficulty traces of blood continue to appear in the expectoration and not infrequently small fragments of tumor tissue or the products of inflammation.

Certain patients, especially after bouts of coughing, complain of repeated painful, sometimes malodorous eructations of air which has been forced from the air passages into the esophagus. Others experience vomiting. Occasionally subcutaneous emphysema is observed in the neck as a result of the escape of air into the mediastinum. Another result of this free intercommunication sometimes experienced is a fall of the pressure within the trachea and larynx caused by the sudden rush of air into the esophagus, leading to an alteration of the voice from failure of the vocal cords to vibrate. Aphonia may be the result.

Usually the acuteness of the symptoms subsides rather quickly. It seems that a certain degree of tolerance becomes established which, although it does not prevent the ejection of food which has escaped into the trachea or bronchial lumen, appears to diminish the cough-producing reflex activity of the mucous membranes.

It is during this phase, also, that the traumatized inflamed respiratory epithelium loses its defense mechanism against the infection, which gradually reaches the terminal bronchi and the lung parenchyma. The purely mechanical aspects of the condition then become less predominant and the pulmonary complications take over.

3 FINAL STAGE The course of the condition ends with the development of bronchopulmonary suppuration. The spread of infection usually results from minute traumatisms and irritation of the mucosa. It is probable, also, that the lodgement of small particles of food in the peripheral bronchi is responsible for a large part of the difficulty by setting up the usual reaction to a foreign body.

These bronchopulmonary complications sometimes take the form of diffuse pulmonary suppuration, sometimes that of bronchopneumonia. Less often a lung abscess develops or somewhat more frequently the lung becomes gangrenous. The temperature rises and an irregular continuous fever develops. The sputum becomes fetid like that of frank gangrene, or it may assume a fecal odor. The bacterial invaders are varied and numerous without any typical flora. There is usually, however, a predominance of intestinal organisms, especially when the odor is fecal.

The general condition deteriorates rapidly. Malnutrition progresses at an accelerating pace as the patient becomes more unwilling to eat until finally a gastrostomy has to be performed. This procedure, performed as it is upon an individual already profoundly debilitated, takes on a particular degree of seriousness.

In over two-thirds of the patients, death results from the pulmonary complications. In the type of case described, survival after the first characteristic signs rarely exceeds one month or less. A longer interval, up to six months as reported by Steiner, is exceptional. Death may occur, however, much more swiftly and unexpectedly as a result of an unusually violent attack of suffocation or of a massive hemorrhage which inundates the lungs.

In a few patients, death follows the expected evolution of the underlying causal disease.

A special type of case which deserves better recognition occurs in patients who for many months have had an inflying tube because of an esophageal stricture. The esophagus no longer serves any purpose but the passage of saliva.

These patients do not have easily recognizable regurgitation of food, but they experience a frequent, almost continuous coughing which produces only clear sometimes stringy and frothy sputum, actually almost pure saliva. Often with children one is surprised to learn that they expectorate, although the broncho-pulmonary examination reveals nothing particular but some moist rales over the lung fields. This simple sign seems to be very characteristic and has made it possible several times to suspect the presence of a fistula before it is revealed by the roentgen examination which serves to confirm the clinical impression.

Roentgen Examination

Fluoroscopic examination before the ingestion of opaque mixture often reveals the presence of shadows due to bronchial inflammation or foci of pulmonary involvement which have a value when it comes to the localization of the fistula. The barium is then administered and its passage through the esophagus is observed. In addition to the obvious evidences of stenosis prestrictural dila-



FIGURE 296 Roentgenogram showing filling of bronchi and trachea after deglutition of barium mixture. Note evidence of bronchiectasis (right middle and left lower lobes) (Dr Sarbazin.)

tation, and so forth, the opaque liquid can be observed in its usually, rather short passage into the trachea or the bronchi, sometimes at two or even several points. At this moment, bouts of coughing bring about the dispersion and ejection of the liquid. If the material is allowed to trickle down slowly in small amounts, however, the defensive mucosal reflex may be controlled to some extent, where upon the liquid may be seen to fill the bronchi as well as might be accomplished by the usual endotracheal Lipiodol injection intended to produce a bronchogram. A film made at that moment gives a perfect demonstration of the location characteristics, and importance of the communication (Fig. 296).

When the fistula is a small one, it may be necessary to place the patient in various positions in order to demonstrate it. Actually it is preferable in all cases to have the patient swallow small amounts of Lipiodol instead of the usual barium mixture.

Endoscopy

In the absence of an etiologic diagnosis, it is most important to begin with an esophagoscopy because the esophagus is the usual site of the initial lesion. This examination makes it possible to establish a diagnosis of this underlying pathology often by means of a biopsy, and to study various aspects of the perforation.

As a test of the presence of a fistula one may insufflate through the esophagoscope a colored gas or tobacco smoke which the patient then eliminates by way of the larynx during exhalation. This technique is of special value with minute openings where the diagnosis remains uncertain.

During the same sitting also it is possible to perform a tracheobronchoscopy without much inconvenience. In those patients, however, who are cachectic it is more prudent to wait twenty-four to forty-eight hours. At this examination usually a small bulging prominence is seen, the exploration of which may cause bleeding and from which in favorable cases a clear, thin fluid recognizable as saliva may be seen to exude. Sometimes when the esophagoscopy may have been inconclusive as to the place of origin of the communication, a biopsy may ultimately establish the respiratory tract origin of the neoplasm. There is no doubt, however, when the opening is exceptionally large, that these explorations lose much of their value. On the other hand, in the latent or unrecognized forms of the condition they make it possible to establish the diagnosis and contribute greatly to the localization of the exact site of the fistula.

Differential Diagnosis

When dealing with a patient who is already under observation and on whom a diagnosis of esophageal disease has already been made, the clinical signs and the investigations enumerated above provide evidence conclusive enough to eliminate any other syndrome. Occasions arise, however, when the causative disturbance is unrecognized or still obscure, in which circumstance the perforation may present under the guise of a pulmonary suppuration whose etiology must be sought. Or the premonitory signs such as cough and hemoptysis lead

to a presumptive diagnosis of incipient tuberculosis until the time when the characteristic syndrome becomes manifest

As a matter of fact, there is no differential diagnosis, properly speaking

There are, however, cases seen during the course of a fluoroscopic examination in which, after the passage of a barium mixture through the esophagus, traces of it appear in the respiratory tract. This may cause a deceiving picture. Several such cases have already been described in the study of cancer of the upper orifice or of the middle third of the esophagus and also in various forms of esophageal dyskinesia. These 'pseudo fistulae' are actually the result of laryngeal spillover. All these patients have recurrent nerve involvement which hinders the physiological closing of the larynx during the process of deglutition.

In the differential diagnosis of the underlying cause of the fistula the variable manner in which the condition appears is important. For example, the patient may be under observation or treatment for a condition already known. The syndrome caused by a perforation is then too obvious to pass unnoticed. It is recognized at once and its true cause is established. There are, however, instances when the perforation reveals itself only after it is actually completely formed, or it remains unrecognized or is found only at autopsy, such as may happen in a patient on whom a gastrostomy was performed because of a carcinoma. In such a patient the passage of material through the esophagus is reduced to a minimum and the swallowing of saliva is not always sufficient to give rise to the characteristic bouts of coughing.

In another situation the syndrome becomes established in a patient not yet under treatment and often in apparent good health. With these except for the rare cases of traumatism in which the etiology is evident it is necessary at first to center one's observations on the esophagus. The incipient signs of the esophageal syndrome should be searched for, such as salivation, slight and more or less erratic thoracic pains, and immediate regurgitation of food swallowed.

The knowledge of previous deglutition of a caustic liquid resulting in stenosis sometimes facilitates the search.

More often the absence of any previous esophageal difficulty, the sex (men above all), the age, the early deterioration of the general condition, and the rapid loss of weight will direct attention toward neoplasm, the existence of which is confirmed by an esophagoscopy aided by a biopsy.

As an unusual circumstance esophagoscopy may disclose the presence of an unrecognized perforating foreign body. Likewise the previous history of pain, variable spasmodic stenosis, and esophagoscopic evidence of the lesion permit the diagnosis of a perforated ulcer of the esophagus. This searching of the esophagus, however, may lead to nothing. Former bronchopulmonary difficulties, slight pulmonary troubles, exertional dyspnea, paroxysmal coughing, and slight hemoptysis should lead to the performance of a tracheobronchoscopy after suitable roentgen studies have been made.

This examination may reveal a more or less proliferative tumor, the histological study of which confirms the diagnosis of carcinoma. However, nothing but the bronchial orifice of the fistula may be seen at the very time when the thickening of the tracheal or bronchial wall and the considerable widening of the tracheal carina suggest the presence of an adenopathy. In these patients

DISEASES OF THE ESOPHAGUS

roentgen examination will have already demonstrated more or less voluminous mediastinal, hilar, and peritracheal or bronchial shadows. The previous history may suggest tuberculosis. There is then the question of a suppurative adenitis which has ended in a fistula between the respiratory tract and the esophagus. Syphilitic gummas, tuberculous ulcers, and other more unusual causes will be diagnosed less readily by their special characteristics than by the exclusion of all other predisposing causes.

Prognosis

It must be recognized that the prognosis of fistulous communications between the esophagus and the respiratory tract is exceptionally grave, as much because of the inherent nature of the complication itself as because of the seriousness of the underlying disease. In fact, it must not be forgotten that approximately 50 to 60 per cent of the perforations occur in the course of a carcinoma of the esophagus, in addition to those occurring in advanced pulmonary tuberculosis complicated by suppurative mediastinal lymphadenitis. The others are chiefly due to tracheal or bronchial neoplasms. On the other hand, in a young subject, provided a gastrostomy has been performed previously, a fistula is often well tolerated and causes only a minimum of discomforts.

There are also fistulae of a non-neoplastic origin which continue to be well tolerated indefinitely or even heal as a result of appropriate treatment. These are all small perforations due to nonprogressive or curable conditions. Reported cases include fistulae due to periesophageal abscess, foreign bodies, syphilis, pulmonary abscess, and acute corrosive esophagitis.

Of a total of 104 patients with fistulae from all causes whose cases were followed thoroughly by A. Barrut, ninety-two died within a comparatively brief time, seven survived the fistula and five were lost to follow-up.

Treatment

When one reviews the literature regarding esophagorespiratory fistulae, one is struck with the uncertainties of the treatment. In certain types of case, several precautions may be taken to avoid as much as possible the establishment of a communication. Once it is established, however, there is little to be done unless it is a benign curable fistula.

In any patient who might develop such a fistula, it is recommended by way of prevention to refrain from any endoscopic manipulations or at least to reduce them to a minimum. All the more important is it to keep in mind the possibility when there are already warning signs of tracheobronchial irritation such as paroxysmal cough and hemoptysis. Esophagoscopy, therefore, and especially blind bougienage, even if fluoroscopic guidance is used, should be proscribed. Oral feeding must be curtailed and a gastrostomy may be performed.

The esophagus can also be intubated, as might be done once the presence of a fistula is confirmed. In spite of everything the possibility of avoiding a fistula is remote.

In the presence of an established fistula as revealed by the characteristic

symptomatology, two situations must be considered. In the first the fistula is the result of a condition which has a probably hopeless prognosis. In this instance the occurrence of a fistula is a complication which hastens the approaching fatal termination. There is no treatment with which to combat it except palliatives such as gastrostomy, provided as in patients with carcinoma of the esophagus, this has not been done already. The insertion of a rubber tube in the absence of a stenosis is another alternative. Contrary to expectations, this may be tolerated for a fairly long period of time and may provide a measure of relief from distress by making feeding by gavage possible.

When the fistula is caused by relatively benign causes, the situation is different. Here specific treatment may be applied according to the etiology of the condition. A foreign body may be extracted, allowing the fistula to heal or antisyphilitic treatment may be administered if that disease is at fault. Furthermore in this type of case local applications may sometimes be helpful. These include the application of zinc chloride or weak solutions of silver nitrate.

In addition to these measures which must be carried out with care there is general agreement that swallowing must be forbidden. Even the saliva should be expectorated and a gastrostomy must be established to provide proper alimentation as well as to put the esophagus at rest.

The prospects of success are not great but the results are not universally futile as in the preceding group.

When it comes to intercommunicating perforations often of indeterminate cause which do not tend to increase in size they may advantageously be treated by repeated applications of caustic agents by way of both the esophagus and the trachea alternately.

Lastly, it should be pointed out that in many instances of nonmalignant fistula between the esophagus and the air passageways it is much better to resort to early surgical intervention on the fistula itself than to wait for improvement to follow the inadequate measures just described or the natural tendency

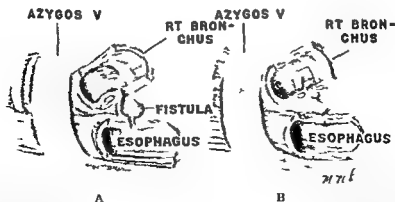


FIGURE 297 Drawing made at operation for the closure of esophagobronchial fistula (same case as Figure 219 page 273) A shows the relative size and shape of the fistula tract B the appearance of the bronchus and esophagus after excision of the tract and closure of each end.

DISEASES OF THE ESOPHAGUS

of the process to heal. Not infrequently if this is done before there is too great an amount of inflammatory reaction, the fistula may be closed and a resection either of a portion of the lung or of the esophagus may thereby be avoided (Fig 297). Even if a resection should be required, the immediate improvement which can be expected makes it worth while to assume the relatively small additional risk involved.

CHAPTER 22

Rupture and External Injury

Rupture

Rupture Due to External Violence

The esophagus is a well protected and highly mobile organ. Except for injuries of exceptional violence when the thoracic cage is badly damaged, it is rarely disturbed as a result of the application of external force. The type of accident which may bring this about is likely to be that where the chest is crushed, as between two moving vehicles, when the patient is pinned against a wall by an automobile or a wagon or when a car falls upon a person who may be lying beneath it. Sometimes a jet of compressed air over the open mouth may cause an esophageal rupture. Falls from airplanes are another cause. During the last world war ruptures of the esophagus were observed after the explosion of bombs, especially when the subject had his mouth open.

Usually the esophageal rupture is discovered only at autopsy. The pathogenesis has been explained usually on the assumption that the esophagus was torn by the direct application of force. It is possible to conceive also, especially with the body flexed, that the esophagus when compressed at one or more points might form a sort of balloon, the walls of which must yield to the sudden increase of pressure within and become torn by a bursting force. This is the more likely mechanism. The signs of shock and the evidences of abdominal injury often predominate and obscure the clinical picture.

The PROGNOSIS is usually bad although, since the employment of antibiotics, some recoveries have been observed.

Spontaneous Rupture

This term is actually a misnomer because the rupture always occurs as a result of the application of force even though not external.

MECHANISM OF OCCURRENCE. In general all the case reports since the original observation on the Dutch admiral published by Boerhaave in 1724 have followed the same pattern. The accident may occur as the result of violent

DISEASES OF THE ESOPHAGUS

efforts to vomit following the taking of an emetic, or after a sudden severe exertion such as a wrestling match indulged in following the ingestion of a large meal or copious drinking. Rupture has been observed, also during attempts to lavage the stomach, during parturition, or during the forceful vomiting in certain neurological conditions such as meningitis or central nervous system syphilis.

Certain experimenters, notably Zimmsen, MacKenzie, A. L. Taylor, and Marian, have attempted to calculate the force necessary to bring about a rupture of the walls of the esophagus. The reports vary from 2.5 to 10 kg. At the lower end where the wall appears to be more fragile, rupture of a healthy esophagus may follow the application of 0.5 to 1.5 kg. pressure. These pressures are disproportionate with that of a vomiting effort or even in the case of Morley, with that resulting from a simple jump from a carriage to the ground. It has been observed that spontaneous ruptures are particularly frequent in alcoholics who are afflicted with chronic esophagitis (Walker reports 14 alcoholics out of 22 patients). Other conditions in which a predisposition has been cited are cerebral infections, active peptic ulcerations of the mucosa, and healed ulcers covered by a smooth atrophic epithelium. Inasmuch as the occurrence takes place near the cardia, which is a transitional zone the mucosa of which is rich in acid pepsin producing glands, the pathogenesis may in the last analysis be considered more like that of gastric ulcer, which is often complicated by a perforation.

Furthermore, several histological studies have disclosed a regional disappearance of the esophageal epithelium with an infiltration by lymphocytes distributed throughout the muscle fibers, the formation of microscopic abscesses in the submucosa and chorion, obliterative endarteritis, and the presence of bacteria or spirochetes.

Other theories assume the presence of esophagomalacia (nutritional insufficiency of the esophagus), varices, atonic dilatation, and bacterial infarction.

NATURE OF INJURY. Postmortem examination has shown that the site of rupture is usually immediately above or below the diaphragm. Viewed from within, the rupture usually appears to be linear, clean, complete, and as though made by a knife cut. All coats of the esophagus are involved in a longitudinal direction over an extent of 1 to 8 or more centimeters. The posterior and left lateral wall of the organ is most often involved. A rupture of the anterior surface and right side is more unusual. In the case described by Boerhaave, the rupture was circular, and transverse tears have been reported by others. In a single case reported by Allen the break occurred as high as the tracheal bifurcation.

The reason for the left posterior location of the majority of ruptures is not clear, but it has been proposed by Beal that during vomiting the rupture results from violent propulsion of the gastric contents against the posterior surface of the esophagus, a region where resistance is weak.

CLINICAL CHARACTERISTICS. The patient suddenly experiences an excruciatingly painful deep, tearing sensation in the thorax, the epigastrium or in the lumbar region. Immediate syncope often occurs and the patient rapidly enters a state of shock. The face becomes pale, the pupils are dilated, the pulse is small and rapid, the respirations are shallow, the extremities grow cold, intense sweating occurs, and thirst ensues. The pain is exquisite.

Local examination may be unrevealing or it may show left upper quadrant abdominal tenderness and muscle spasm

In about one half of the patients a characteristic sign develops rapidly. This is subcutaneous emphysema at the base of the neck which spreads quickly, infiltrating the mediastinum, the skin of the thorax, the face, and the limbs. Auscultation reveals the presence of fine rales at the bases of the lungs, especially the left, and eventually the signs of hydropneumothorax. Exploratory thoracentesis may reveal fluid having an acrid odor with all the gross characteristics of gastric juice or even containing alimentary debris. Sometimes there is a brownish or sanguinolent exudative fluid.

The condition worsens rapidly from hour to hour. The patient grows cyanotic, begins to suffocate, and, if surgical intervention is not practiced, promptly dies, often within twenty-four to thirty-six hours from the onset of the injury.

DIFFERENTIAL DIAGNOSIS Because of the frequent presence of left upper quadrant tenderness and muscle spasm, a perforated gastric or duodenal ulcer, acute hemorrhagic pancreatitis, mesenteric thrombosis or dissecting aneurysm of the aorta may be thought of. An attack of coronary thrombosis or of spontaneous pneumothorax may be suspected.

The roentgen film of the chest usually shows emphysema and a fluid level in the mediastinum with a left pneumothorax. These findings should help to exclude most of the aforesaid diagnoses. The absence of a collection of air beneath the diaphragm is helpful. The finding of food particles or ingested liquids in the material removed by thoracentesis gives valuable evidence.

Although the mistake may be made of performing an exploratory laparotomy, the clinical picture is actually so characteristic that the diagnosis of rupture of the esophagus should be suspected in the majority of instances and the incision made in the chest.

PROGNOSIS Without surgical intervention the prognosis is grave. The rupture tends to enlarge and death may ensue in twenty-four to forty-eight hours. In some instances a fatal termination may occur with astonishing rapidity (four to six hours).

If the perforation is not extensive, more like a simple fissure, death occurs more slowly but rarely later than one week after the injury. In such patients the evidences of overwhelming sepsis predominate. The infection often involves the pleura soon after the accident and irritation produced by the gastric contents evokes a purulent mediastinitis and empyema.

TREATMENT Prior to 1940 the situation was practically hopeless. Since that time, however, a number of cures have been reported. The success of the treatment depends directly upon the early establishment of a correct diagnosis and the prompt resort to surgery. Necessary measures to overcome the effects of shock must be employed during the short time which may be allowed for the preliminary preparation of the patient. Intravenous fluids and blood transfusions are administered as indicated. Antibiotics are started by the intravenous route. If there is evidence of tension pneumothorax, relief is obtained by needle aspiration.

The operation should be performed with endotracheal anesthesia using a

standard thoracotomy incision, usually through the left eighth intercostal space. The fluid is evacuated from the pleural cavity by aspiration. The lower esophagus is mobilized without sacrifice of its blood supply and the rent is disclosed. Interrupted stitches of fine silk or catgut are employed to suture the defect as in any esophagotomy or anastomosis. A catheter is inserted to drain the pleural space and the wound is closed in the usual manner. Continuous aspiration of the tube is maintained for several days.

The *aftercare* includes the continuance of large doses of antibiotics such as Chloromycetin or Achromycin, which may be employed parenterally, and of parenteral alimentation. Oral intake is avoided for twenty-four to forty-eight hours, after which small amounts of fluid may be started. If the improvement of the patient continues, the remainder of the convalescence is to be managed as it would be after an esophagectomy.

Provided that the diagnosis was made early (within twelve hours at the most), an uneventful recovery may be anticipated in the majority of instances.

In cases of longer standing the infection may have become so well established that mediastinal abscesses and empyema may occur as complications after the operation. Disruption of the suture line is then a frequent occurrence. In the treatment of this complication the pleural cavity must be drained by rib resection and a gastrostomy performed to provide for the patient's nutrition. If the infection is then treated intensively, the patient may recover, but an esophagopleurocutaneous fistula usually remains.

Four to six months later, depending upon the time required for clearing of the empyema and mediastinitis, the patient may be reoperated upon. It is almost always necessary to perform an esophagectomy, cutting across the esophagus through normal tissues above the fistula opening. Restoration of continuity is performed by esophagogastrostomy.

In the occasional patient who may survive several days of neglect, a definitive operation should not be attempted. The empyema should be drained and a gastrostomy established, after which the case is handled as outlined above.

In a few instances of only slight tearing of the esophagus, the resort to antibiotic medication, proscriptio*n* of swallowing, and the use of parenteral feeding may bring about spontaneous healing of the rupture. With few exceptions, however, it is unwise to embark on such a course because of the impossibility of making certain that the break is small.

External Injuries

Injuries of the Cervical Esophagus

In civilian practice, wounds of this portion of the esophagus are seen almost always in suicides or in victims of crimes of violence as a result of cutting with a razor or a sharp knife. In suicidal lacerations the cut is usually transverse with a slight obliquity, and rather high involving the thyroid membrane, more rarely the cricothyroid membrane. Lower down the trachea is sectioned first and if the instrument is applied with great force, the esophagus may be reached. There is usually a more or less deep involvement of the muscular coat of the

anterior wall without involvement of the mucosa. Complete division is unusual. Because of the fact that the majority of people are right handed the cut is deeper on the left side than on the right. If the direction of the wounding instrument is lateral, the sternocleidomastoid muscle is divided first, then the vessels and the nerves and finally, in the depths of the wound the esophagus.

Wounds of the esophagus alone by pointed objects may be seen but usually they are complicated by lacerations of neighboring organs. Firearm injuries of the esophagus are rare since the neck is a small target.

Projectiles may cause a variety of injuries. There may be only a simple contusion. Sometimes a unilateral perforation is seen which is often rapidly occluded by mucosal herniation, as in intestinal perforations of this nature. In these the projectile may drop into the lumen of the organ from which it is expelled by vomiting or evacuated with the stools. Sometimes a transfixing wound occurs attended by perforation of both walls of the esophagus.

CLINICAL CHARACTERISTICS. Because of the involvement of other organs, the esophageal symptoms and signs are frequently obscured and the injury is discovered only at the time of debridement of the wound or when there are complications. If the wound is large, the esophageal laceration is manifested by painful dysphagia and especially by the escape of saliva and ingested liquids.

With a small injury subcutaneous emphysema induced and aggravated by the movements of deglutition is a helpful sign, though inconstant. Bleeding from the esophagus itself is rarely pronounced.

Infection constitutes the chief danger in wounds of the esophagus. It may develop early, often by the end of twenty-four hours, or it may be delayed, appearing at the time of separation of the eschar or even after the dislodgement of a projectile caught in the esophagus. Less often it results from secondary perforation due to the erosion of the wall of the esophagus by a projectile lying against it or to the rupture of a cervical abscess into the organ. There is always a mixture of organisms present including anaerobes, the toxicity of which may be severe.

Fever appears and the dysphagia increases. A foul fluid exudes from the wound. The tissues become distended. The neck grows larger and becomes indurated. Little by little the symptoms of diffuse phlegmonous esophagitis or of a localized periesophageal abscess develop. The fever is not always high but this may belie the seriousness of the condition. The patient becomes apprehensive. The pulse is small and rapid. Sometimes the fever rises precipitously, the pulse rate climbs, the patient experiences rigors and the breath becomes fetid.

The base of the neck enlarges and the tissues become edematous. Movements are difficult and motion of the trachea is painful. Palpation reveals points of deep tenderness along the trachea. The laryngeal mirror discloses edema of the arytenoids or a swelling of the posterior wall of the pharynx. The clinical picture is that of the evolution of a periesophageal abscess or phlegmon.

DIFFERENTIAL DIAGNOSIS. Save for the obvious cases, the diagnosis is always difficult because some of the classical signs of wounds of the esophagus, notably the subcutaneous emphysema and the dyspnea, may occur with injuries of the air passages.

Esophagoscopy may be employed but is of limited value and must be done very carefully to avoid augmenting the injury.

Fluoroscopic examination of the esophagus after a swallow of barium mixture should not be necessary and is inadvisable.

COMPLICATIONS include mediastinal infection, pyopneumothorax, empyema, purulent pericarditis, hemorrhages, cutaneous fistulae, and esophago-tracheal fistulae.

PROGNOSIS : The prognosis is always serious. It depends upon the rapidity with which surgical intervention is carried out and on the gravity of the associated injuries. In large transecting wounds of the esophagus the inferior end may retract into the mediastinum and the resulting infection, if nothing is done to correct the situation, is usually lethal. Large incomplete transections produced by cutting instruments become complicated by fistulae and cicatricial stenoses. The prognosis in this type of wound of the esophagus must be guarded in the majority of instances.

In wounds produced by firearms, the esophageal lesion may heal spontaneously without serious sequelae. This is especially true in the case of injuries caused by bullets. The most that is usually seen is slight cicatricial narrowing or spastic affections of the mouth of the esophagus caused by irritation and reflex activity of the innervation as a result of trauma occurring in the area. These conditions are easily overcome by simple dilatation. There are usually no serious consequences, but cicatricial stenosis or eccentric web formation at the mouth of the esophagus may be the end result.

Fistulae are not often observed in injuries caused by firearms. They are more likely to succeed large injuries. When there is simultaneous wounding of both the esophagus and the trachea, a separate fistula in each of these organs may occur.

Extensive injuries often heal. Actually they are more likely to heal than the smaller wounds, especially those on the posterior surface of the esophagus. Among the various explanations proposed to account for this fact, other than the ease with which the diagnosis is made so that treatment may be begun earlier, the most likely is that drainage is more free. Furthermore, the seriousness of the prognosis depends on the accompanying injuries of the neighboring organs. Combined injuries of the larynx and the esophagus are the most serious of all.

TREATMENT In every wound of the esophagus including those made by pointed instruments, immediate external intervention is the best means of preventing infection. In the case of a single wound, even if very limited in extent, cleansing and debridement including extraction of projectiles or other foreign bodies and freeing of the esophagus must be practiced. With a verified or suspected lateral injury, as might be caused by a projectile, both sides of the esophagus should be explored and freed. The superior mediastinum should then be walled off by the insertion of gauze packing and wide open drainage of the neck provided.

There is some disagreement regarding the suturing of the esophagus. If the injury is treated early and the tissues are relatively clean, it is undoubtedly wise to close the opening if possible with interrupted sutures of fine catgut.

This prevents the escape of saliva and the enlargement of the wound. On the other hand, in neglected patients or those for any reason seen long after the injury, the tissues are not suitable and infection may be widespread. In these, exteriorization is preferable and closure must be delayed for a later date.

If the esophagus has been cut completely across, the escape of the lower end into the mediastinum must be prevented at all cost. When the patient is seen soon after the injury, an end-to-end suture can usually be accomplished. If there has been much delay, it may be necessary to suture the distal end to the skin of the neck, leaving a gauze pack against the proximal end. After the subsidence of the inflammation, the esophagus may be sutured at a second operation.

Esophagoscopy should rarely be used and then only in cases of minor injury and with great caution.

Aftercare. After the operation, oral feeding must be forbidden during the first few days. The usual parenteral methods are employed.

The use of an inlying nasogastric tube is helpful when a fistula is unavoidable, but it should not be inserted until after the lapse of several days, especially when a closure or end-to-end anastomosis of the esophagus has been performed. In these, the success or failure of the suture will be obvious at the expiration of five to ten days. If a fistula develops, the tube may then be inserted to provide a means of feeding the patient and the intravenous infusions can be abandoned.

With extensive injuries involving loss of much of the esophagus, a gastrostomy should be performed. Ultimate reconstructive surgery must be resorted to in these patients. The technical details will depend upon the nature and extent of the injury and vary from case to case.

Injuries of the Thoracic Esophagus

The thoracic segment of the esophagus is even less frequently injured than the cervical portion. Severe injuries of the other mediastinal structures are always associated and dominate the scene. The esophageal wound in the vast majority of instances is discovered only at autopsy. However, rare instances of injury to the esophagus alone are on record. These may be caused by bone fragments from fractured ribs by projectiles or by stabbing with a knife.

The CLINICAL CHARACTERISTICS are those of a chest injury without distinguishing signs to draw attention to the esophagus. Suspicion may be aroused, however, by the occurrence of violent chest pain during swallowing, especially of liquids, and during coughing. There is often intense thirst and persistent hiccupping.

DIFFERENTIAL DIAGNOSIS is difficult because of the predominance of the evidences of injury to other organs. The most helpful signs are regurgitation and vomiting of bloody material, cervical emphysema, and retrosternal tympany. The latter signs are more characteristic of lacerations of the trachea and bronchi.

PROGNOSIS. The prognosis is grave because of mediastinal infection, which has ample opportunity to develop owing to the infrequency of an early diagnosis of esophageal injury in these patients. Under such circumstances it is easy to understand the frequency of a fatal outcome in this type of injury after several hours or days of dyspnea, thoracic pain, and fever. Death is usually due to purulent mediastinitis.

The only hope is the adoption of a more aggressive approach in the treatment of all patients with a penetrating injury to the chest

TREATMENT : If patients are seen soon after sustaining a penetrating injury of the thorax, it is imperative to perform an exploratory thoracotomy whenever suspicion of esophageal injury is aroused. If this is not done, and in all patients seen late after the injury, the problem evolves into one of treating suppurative mediastinitis in the patients who survive the first few days.

Posterior mediastinotomy must be employed to drain accumulations of pus, which can be detected by a widened mediastinal shadow in the roentgen film. An esophagocutaneous fistula is the almost inevitable sequel of this exteriorization of the infection. A gastrostomy must then be performed and oral intake restricted.

As a final measure in those who survive the mediastinal sepsis, a direct attack must almost always be made. It is usually found that resection of the damaged segment is necessary to effect a cure.

Liberal utilization of appropriate antibiotics is a valuable adjunct to the surgical measures which must be employed according to accepted principles of technique.

The employment of the antibiotics has greatly altered the gravity of these injuries and because of recent progress in the treatment of shock and hemorrhage and in esophageal surgery, a large proportion of patients with wounds of the esophagus can be saved who would formerly have been doomed.

CHAPTER 23

Instrumental Perforations (Endoscopic Injuries, Etc)

The esophagus is the most intolerant of all the organs. It is a contaminated tract containing a bacterial flora rich in harmful forms, above all the anaerobes. Its walls are thin and fragile. It has no serous coat, its blood supply is often tenuous. Furthermore, it occupies the middle of the mediastinum where, in case of perforation, dissemination of infection throughout the mediastinal connective tissue is the inevitable and often fatal result. Finally, the retrovisceral (prevertebral) fascial space in the neck communicates directly with the cellular tissues of the posterior mediastinum and an infection, once it has spread downward, meets no obstacle capable of preventing its widespread diffusion.

Etiology and Pathogenesis

The usual causes of instrumental perforation are efforts to extract a foreign body, bougienage, esophagoscopy with or without biopsy, and the passage of a gastroscope. Abnormal practices on the part of the insane involving the swallowing of metallic objects or an accident to a sword swallower are examples of occasional causes.

EFFORTS TO EXTRACT A FOREIGN BODY LODGED IN THE ESOPHAGUS. These efforts are a frequent source of perforation, especially when they are made by unskilled persons or when improper techniques such as the use of hooks, hemostatic forceps, or other dangerous grasping instruments are employed without the aid of visual control. Too vigorous efforts are often made by the patient himself, who may try to push the object down or pry it loose with crude instruments such as a corset stay.

When it comes to perforations occurring during efforts made by qualified specialists using proper instruments, all sorts of factors may be involved. Avoidance of injury may depend upon the promptness with which the effort to



FIGURE 298 Drawing illustrating perforation by a bougie as it might occur in cases of cicatricial stricture of the esophagus with mediastinal involvement where the esophagus becomes angulated forming thin walled pockets into which the tip of the instrument may pass with resulting perforation. The accident is much less likely to occur if such manipulations are performed under the fluoroscope a precaution which should never be omitted.

extract the object is made, a careful study of the roentgen films, endoscopic visualization of the object, the search for areas where the object is not stuck, the employment of Adrenalin to shrink the tissues, the care and deliberation with which the procedure is carried out, and other fundamental precepts. In all neglected cases the occurrence of perforations may be unavoidable, even in expert hands. Finally, before any effort to extract a foreign body is made, it is important to be sure that there is not a perforation already present, made by the object itself. All these matters are enlarged upon in Chapter 24 dealing with foreign bodies of the esophagus.

PERFORATIONS DURING BOUGIEVAGE When the esophagus is healthy, a pliable rubber bougie or tube manipulated with care cannot perforate the esophagus. When the organ is diseased however, this complication may occur especially in the presence of cicatricial stenosis and tumors, more rarely in benign ulcers or in tuberculosis or syphilis.

In cicatricial stenoses the danger is always menacing, even in the hands of the most expert. This is true for several reasons. The wall of the esophagus is thin. It is often friable and cracks or tears easily. Because of mediastinal adhesions, there are often angulations which fix the organ in an abnormal fashion and lead to sacculations in which the bougie may become engaged to cause a perforation (Fig. 298). With the blind techniques formerly employed, the patient usually succumbed sooner or later as a result of perforation and the danger bore a direct relation to the size of the distended esophagus above the stricture.

As with foreign bodies one is horrified by the methods employed by certain persons with old strictures who have attempted to do their own dilatation. The number of perforations in localities where chronic corrosive esophagitis is a common occurrence has increased because of the lack of gum-coated bougies, and several perforations caused by tempting fortune with metallic objects have been observed. For these reasons, self-dilatation is to be condemned.

The placing of a radium-containing sound in a patient with carcinoma of

the esophagus may also provoke a perforation. This technique especially demands a careful study of the lesion and particularly the recognition of the presence of periesophagitis.

PERFORATIONS DURING OBSERVATION ESOPHAGOSCOPY The blind part of every esophagoscopy is the passage of the cricopharyngeal sphincter where there is danger of perforation in the weak zone situated between the oblique and transverse fibers of the inferior constrictor muscle (see Chapter 4).

If the tube is allowed to follow its own course, and if it is advanced forcibly, a perforation is sure to occur in this weak zone toward which the instrument is naturally guided by the muscular structures. If the angle of introduction is wrong or if too much force is used, the esophagoscope will penetrate into the mediastinum without the knowledge of the endoscopist (Fig. 91, page 111).

If a biopsy is required, great caution must be observed and in the dangerous area close to the sphincter the use of a curette is safer than a biopsy forceps.

GASTROSCOPY Perforation of the wall of the esophagus during the introduction of a gastroscope has been reported. The occurrence is unusual. Schindler experienced only four such accidents in over 22,000 examinations. Only one of these was fatal.

Pathological Anatomy

Instrumental perforations are much more frequent in the cervical esophagus and in the portion just above the tracheal bifurcation than in the lower thoracic and abdominal segments. In a group of ninety nine cases from the literature the relative frequency of occurrence at various levels is as follows:

Hypopharynx and high neck	7
Lower neck	70
Upper thorax (above tracheal bifurcation)	12
Lower thorax (below tracheal bifurcation)	10
	<hr/> 99

The nature of the injury is subject to great variations. There may be small erosions of the mucosa which are of little consequence. Punctiform perforations involving the entire thickness of the esophageal wall may occur. These are likely to be unrecognized. Larger injuries involve either a loss of substance with an opening through the mucosa and submucosa having rounded edges, or a longitudinal rent through the wall which when viewed through the esophagoscope looks like a red line or a bleeding groove or furrow. Serious injuries usually involve more or less extensive divulsion of the esophageal wall.

It cannot be too often repeated that, because of the contamination of the esophageal lumen and the weakness of anatomical barriers, even a minimal injury of the esophagus may end in a serious infection. Any injury which breaks the epithelium permits bacterial invasion of the esophageal tissues. The intensity of the resulting infection varies according to the nature of the organism, its virulence and the resistance of the host. The latter factor is very important for, although all patients who have swallowed a bone or a dental prosthesis experience mucosal abrasions, not all of them develop complicating infections. The saliva is full of aerobic pathogens including hemolytic streptococci associated

with various anaerobic organisms. Stasis at the point of injury is favored by the inflammatory edema and also the foreign body itself if it remains lodged in the esophagus. Contact with gastric juices also may give rise to local tissue digestion in the traumatized area which combines with the process of cellular disintegration evoked by the infection to enhance the effects of the injury.

In the neck a perforation may become complicated by osteomyelitis of the cervical spine.

In the thoracic area, vascular complications may occur. Clerf reports the case of a two-year-old child who died after perforation of the posterior wall of the left ventricle.

In the abdominal segment, perforations usually following bougienage may be through either the anterior or the posterior wall. Much depends upon the nature of the lesion which prompts the initiation of the treatment (stenosis, cancer, diverticulum, etc.)

Clinical Characteristics

SYMPTOMS The symptoms vary to some extent, depending upon the location of the perforation unless treatment has been delayed, when an injury located in the cervical area may have given rise to a spread of infection into the mediastinum.

In general with perforations occurring in the region from the mouth of the esophagus to the level of the fourth thoracic vertebra, the characteristic findings are emphysema of the cellular tissues of the neck and face, and pain in the neck, especially at the base, with radiation to the shoulders. Sometimes, even with high perforations, abdominal muscle spasm may be observed. A perforation of the thoracic region below the level of the aortic arch is accompanied by the signs of shock and evidences of infection with interscapular pain and shallow respirations, a rapid pulse, and facial pallor. With perforations of the lower thoracic and abdominal segments, the pain is referred to the epigastric region and abdominal muscle spasm is pronounced. The general signs just mentioned are also striking.

There is a difference, also, depending upon whether the patient is seen early or after a period of delay. The perforation may be sudden as a result of the extraction of a foreign body or after an exploratory esophagoscopy. In either instance the patient experiences acute pain in the base of the neck or chest, immediate painful embarrassment of deglutition, inability to swallow even liquids, sometimes the vomiting of blood, and marked anxiety. Perforations of the thoracic esophagus, where the pain is often intense enough to interfere with the patient's movements, may compel him to walk stooped over and with short steps. Salivation may be abundant and bloody.

The signs of shock develop soon and the picture characteristic of perforation of a hollow viscus ensues. The pain is felt in the neck, the chest, or the abdomen according to the level of the injury. Muscle spasm of the abdomen is an early sign in low perforations.

When the perforation takes place after a latent period, there are always evidences of infection. The usual symptoms are due to esophagitis. There is local pain, and tenderness may be elicited when searched for. Dysphagia de-

velops gradually, the fever rises, the neck grows swollen, and a suppurative periesophagitis develops. Localized abscesses and phlegmons or mediastinitis of variable severity are frequent complications. Deglutition improves as a result of incision and drainage, but a fistula usually follows.

LOCAL PHYSICAL SIGNS Subcutaneous emphysema due to the penetration of air into the loose connective tissue layers is a characteristic sign. It begins in the suprasternal and supraclavicular areas and along the carotid sheaths, whence it spreads throughout the rest of the neck and chest and into the mediastinum. After the development of infection in this emphysematous tissue the gases produced by anaerobic organisms become commingled with the air. The emphysema occurs early, often in a matter of minutes or several hours after the injury. Its presence should give warning of the accident, but it is not always recognized.

Sometimes on exposure of the esophagus, no actual perforation is found. In these instances a superficial abrasion of the mucosa covering the cricoid cartilage suffices to produce the insufflation of air. On the other hand, emphysema may be lacking even when an obvious perforation is recognized.

Careful palpation of the anterolateral regions of the neck below the mandible and along the carotid sheaths should be practiced. If the emphysema is slight the air which infiltrates the cellular spaces may be dispersed with light pressure but as soon as the patient swallows it reappears.

In large clear-cut perforations the air infiltrates the subcutaneous tissues rapidly and little by little the signs of compression develop. When a foreign body is present the emphysema is particularly painful and is complicated by the transudation of a serous fluid which may appear with great rapidity. The sides of the neck become indurated and palpation becomes painful. Movement of the trachea and larynx increases the pain. Tenderness on pressure over the area where a foreign body is lodged however, is not necessarily indicative of the presence of a perforation.

If the perforation is in the broncho-aortic or subaortic zone muscle spasm of the epigastrium though slight at first, can almost always be elicited. As already mentioned this sign may be observed even when the perforation is high in the neck.

Hyper resonance to percussion and the disappearance of the cardiac dullness indicate the presence of mediastinal emphysema either primary as with injuries of the thoracic segment, or secondary to an esophageal perforation in the neck.

Regurgitation of bloody fluid occurs early and continues for some time. It is often abundant.

In perforations of the subdiaphragmatic zone, contracture of the epigastrium is boardlike abdomen and loss of dullness to percussion over the liver are characteristic.

The outstanding clinical manifestation of perforations of the cervical esophagus is subcutaneous emphysema and of perforations in the lower portions, excruciating pain and collapse followed by the development of spasm of the upper abdomen.

GENERAL SIGNS are in the beginning those of shock and later the manifestations of infection. One is impressed by the restlessness of the patient, the increasing dyspnea, the respiratory oppression, the retrosternal and interscapular pain, the rapid elevation of body temperature, disturbances of the pulse, chills, somnolence, and delirium.

The signs of a subphrenic abscess are usually tenderness in the costovertebral angle and induration and tenderness in the flank and lumbar region. However, because such an abscess develops in a less absorbent and less irritable cellular tissue than is the peritoneum, the local symptoms and signs are often insidious and the only evidence at first may be merely the manifestations of toxicity.

Peritonitis may develop but, thanks to the rapid formation of adhesions, the suppuration usually becomes walled off in the upper part of the abdomen above the level of the umbilicus.



FIGURE 299 Roentgen films of the neck of a patient with an esophageal perforation resulting from laceration by the point of an open safety pin lodged in the cervical segment show the Minnigerode sign. A The trachea at A shadows between the arrows represent air in the neck and superior mediastinum. B Lateral view, same patient. Arrows point to a column of air in the retropharyngeal space (Child age 3 years cured after removal of the pin by esophagotomy). C Diagram showing the three possibilities for the presence of air or other gas between the trachea and the vertebral column as seen in lateral roentgen film. 1 foreign body in the esophagus. 2 large bubble of gas indicating the probable presence of a periesophageal abscess with perforation. 3 extensive emphysema perforation is certain (Courtesy of E. Hinzanga).

Roentgen Examination

No one denies the utility of emergency roentgen examination of the acute abdomen when diagnosis is difficult. This is just as imperative when esophageal perforation is suspected as for a perforation of the stomach or duodenum. In fact, the emphysema which is the predominating clinical sign may be recognized at fluoroscopy several hours before it can be elicited by palpation and percussion. Furthermore, a foreign body may be discovered and its position identified.

Films should be taken in all projections (frontal, lateral, and oblique). If a foreign body is implicated, frontal and lateral films should be obtained both before and after its extraction. The examination should be made at first without the use of a swallow of barium.

In the presence of emphysema bubbles of gas in the periesophageal tissues can be detected in the films. This is the so called Minnigerode sign which is of great diagnostic value (Fig. 299). It is characterized at first by the presence in the region between the esophagus and the spine of clearly defined air spaces. Later, a thickening of the posterior wall of the esophagus is seen with or without emphysema. Minnigerode advises that in all patients in whom a perforation of



FIGURE 300 Roentgenogram lateral film after ingestion of Lipiodol (woman aged 64 years). View made 3 days after esophagoscopy for foreign body showing emphysema, a large bubble of air, and extensive retropharyngeal and esophageal abscess. Cure by surgical intervention. (E. Huizinga.)



FIGURE 301 : Roentgen film made after ingestion of Lipiodol (same patient as in Figure 300) This shows the Lipiodol filled periesophageal abscess descending as far as the sixth thoracic vertebra (E. Huizinga)

the esophagus is suspected, especially before or after the extraction of a foreign body or after an esophagoscopy which is complicated by symptoms, roentgen films should be made daily to look for evidence of air extravasation.

In some instances an error of interpretation may be made. For example, when a foreign body has been lodged for a long time in the esophagus, clear spaces occasionally appear in the absence of a perforation. These are due to a swelling of the soft parts in the prevertebral space. The phenomenon subsides spontaneously after the ablation of the foreign body (Fig. 299, C, 1).

E. Huizinga has given a perfect demonstration of the value of the several forms which the air shadow may assume, from the small bubble to the large periesophageal accumulation and finally to the large dissemination characteristic of emphysema which indicates that a free perforation has occurred and demands intervention (Figs. 299 and 300).

Once the existence of air in the periesophageal tissues has been demonstrated, the films are helpful in localizing a foreign body if present, and in deciding whether the object lies within or outside of the esophagus. Films of

the abdomen may disclose the presence of air beneath the diaphragm when the esophageal perforation is in the lower segment

When a perforation is suspected but not proved, a mouthful of barium or Lipiodol may be administered. If a large opening exists, the opaque material will be seen to enter the periesophageal tissues (Fig. 301). Small rents in the esophageal wall may not be seen by this technique. There are objections to the use of the method, however, because of the well known irritating effect of the mixture which may tend to increase the seriousness of the infection. In spite of the diagnostic value and interest of the use of barium, therefore, it is probably wise to abstain from using it as a routine measure in patients with an obvious perforation.

Esophagoscopy

An esophagoscopy may reveal the presence of an impacted foreign body, the location and appearance of the perforation, and the condition of the surrounding mucosa.

In every instance the difficulties must not be ignored. The early appearance of edema may mask the presence both of a foreign body and of the perforation. There is danger also, though to a lesser extent, that the esophagoscope might aggravate the laceration of the esophagus.

Sometimes bubbles of air may be seen to emerge from the perforation during respiratory movements. In large perforations the loose connective tissue of the mediastinum may actually be seen through the opening.

Prognosis

If the perforation is large and if no adhesions have developed, the outlook is grave. On the other hand if the opening is small or if it appears slowly and if adhesions have walled off the periphery, the purulent collection may evacuate into the esophagus and the perforation may seal over spontaneously. The evacuation of an abscess into the mediastinum, however, is always to be feared because of the usually lethal mediastinitis which follows.

The appearance of a high fever is an ill omen. So also is increasing tenderness on palpation over the carotid sheaths.

Signs of toxicity and evidences of septicemia are ominous. They may constitute an indication for operative intervention.

Treatment

The advent of antibiotic medication to combat infection has greatly altered the seriousness of the condition and the method of treatment. In fact, many cases of esophageal perforation which formerly would have been attended by a fulminating infection and a fatal outcome, nowadays can be expected to follow a benign course ending in healing without surgical intervention. In many instances, however, both antibiotic and surgical treatment must be employed. As in all other situations, antibiotics cannot supplant the drainage of abscesses and the removal of offending foreign bodies.

The patient should be confined rigidly to bed. In order to favor the evacuation of the often abundant secretions which fill the pharynx and to prevent the

aspiration of septic liquids into the respiratory tract, it is wise to elevate the foot of the bed. This position may also prevent to some extent the descent of the infection into the mediastinum. Some authors advise the use of the Trendelenburg position.

Treatment should be directed toward (1) *putting the esophagus at rest*, (2) *measures to overcome the infection* (both local and generalized), particularly the prevention of spread from the region of the perforation, and (3) *supportive measures to improve the general condition of the patient*.

MEASURES CALCULATED TO PUT THE ESOPHAGUS AT REST The esophagus must be put at rest by the interdiction of all oral intake, both liquid and solid. Salivation and esophageal peristalsis may both be counteracted by the administration of atropine in doses of 0.4 to 0.6 mg. Hydration of the patient is maintained by giving intravenous infusions of dextrose in water or salt solution to which vitamins have been added in appropriate amounts. As a temporary measure, a continuous rectal drip may be helpful in maintaining an adequate fluid intake without overuse of the veins.

Certain authors advise immediate performance of a gastrostomy not only to put the injured part at rest but also to provide certain additional advantages. These include the assurance of a means of feeding the patient for a prolonged period of time, avoidance of the regurgitation of irritating gastric juices which takes place around an indwelling nasogastric tube (Levin), provision for administering antibiotic and other medicaments which are usually given by mouth, and creation of the means by which dilatation can be carried out by the continuous thread method.

An alternative procedure which may make it possible to avoid performing a gastrostomy is the insertion of a nasogastric tube through which a nutritious liquid formula can be given (see Appendix). The disadvantage of this technique mentioned above may be overcome by using a tube with an additional lumen which opens into the esophagus for the purpose of continuous aspiration of saliva or fluid regurgitated from the stomach.

MEASURES TO COMBAT INFECTION Since the advent of the antibiotics, one is no longer forced to stand by during the anguishing drama which terminated in the rapid death of the patient overcome by the fulminating toxemia which was so very obvious but impossible to treat. Medication should be started promptly, as soon as the perforation is recognized. Although certain strains of staphylococci have become resistant to it, penicillin should be administered because it is still effective against the formidable hemolytic streptococcus. In addition it is wise to give streptomycin and Chloromycetin, Achromycin, or one of the other broad-spectrum agents, with the usual supervision and precautions. Because of the probability that a mixture of virulent organisms both aerobic and anaerobic, must be dealt with the dosage should be large. The treatment should be pursued long enough to make certain that the danger is past. It should then be omitted abruptly and the patient kept under observation for several days to detect the presence of any lurking localized infection which may have been masked by the effect of the medication.

So far as local treatment is concerned, there is little that can be accomplished. Oral administration of penicillin or streptomycin to secure a direct

effect may have some theoretical value but is not recommended because of the necessity for avoiding the passage of anything through the esophagus. The antibiotics exert their greatest effect when carried into the tissues by the blood stream.

SURGICAL INTERVENTION A surgical attack upon the perforation should be undertaken with two objectives: first, to repair the injury if the patient is seen soon after its occurrence and secondly to provide adequate drainage for accumulations of pus if the condition is not recognized until complicating infection has occurred or if the efforts to prevent infection have been unsuccessful. The approach of course varies depending upon the area of involvement. Endotracheal closed circuit anesthesia should be employed.

Perforations of the cervical and upper mediastinal segments may be reached through a long incision along the anterior margin of the sternocleidomastoid muscle extending from the manubrium below to the level of the thyroid cartilage above. The left side is chosen because of the greater accessibility of the esophagus on that side and the fact that the left recurrent laryngeal nerve can be avoided more easily than the right.

The carotid sheath is retracted and the body of the omohyoid muscle is cut across. The left lobe of the thyroid gland is drawn forward with a traction suture. Several veins and the inferior thyroid artery are divided and ligated. With the aid of a finger introduced behind the esophagus, the entire cervical and upper mediastinal portions of the esophagus may be freed (Fig. 302). (For details of technique see Chapter 25.)

With this exposure any foreign body can be seen and removed, the per-

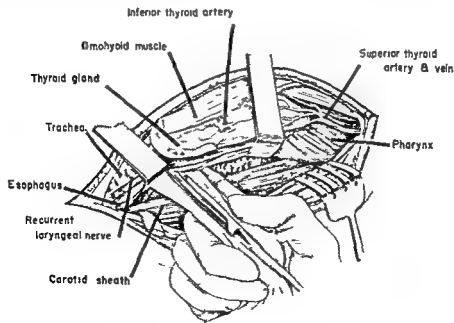


FIGURE 302 Drawing made to show the exposure of the cervical esophagus and prevertebral fascial space for esophagotomy and drainage of retropharyngeal periesophageal and superior mediastinal abscess. (After Piquet.)

foration can be identified, and any deep abscess which may be present can be drained. The exact procedure to be carried out depends upon the actual findings and the length of time between the injury and the operation. A perforation recognized early may be closed with interrupted stitches of fine silk or catgut. If too much time has elapsed before the operation is performed, the edges of the perforation are likely to be necrotic and a proper suture becomes impossible. In this instance it may be necessary to resort to wide open drainage only. If the injury is in the superior mediastinum below the base of the neck, the drains should be placed deep enough to reach a point just below it. By following the posterior surface of the esophagus downward it is possible to reach as low as the fourth dorsal vertebra. Any collection of pus below this level is better drained through a thoracic approach.

The gauze used to wall off the mediastinum should be placed below the perforation to isolate the cellular spaces beside and behind the esophagus which communicate with the neck and the chest. Rubber drains are inserted in the center of the gauze. These must be soft and in no contact with the great vessels in the carotid sheath in order to avoid erosion and hemorrhage.

If there is a wide lateral spread of the infection, it may be wise to make a counter-incision in the right side and insert drains from that side in addition to those on the left.

In patients who have an obvious abscess confined to the superior mediastinum, however, the cervical approach may be unwise because of the risk of dissemination of the infection in the uninvolved fascial spaces of the neck. Furthermore, drainage of the area by this approach is indirect, and efforts to improve it by elevating the foot of the bed to make the head lower than the body or the use of continuous suction on a drainage tube are cumbersome and often uncomfortable for the patient.

During the postoperative period the gauze sometimes used to wall off the mediastinum should be removed gradually, starting on the fourth postoperative day.

The complication most to be feared, in spite of adequate drainage through the neck, is spreading mediastinitis. Nowadays, however, under the influence of antibiotic medication, localized mediastinal abscesses are seen more often than disseminated infection. This complication is nevertheless the most frequent cause of death and may occur even though supposedly adequate drainage has been provided.

The patient should be watched carefully with repeated chest films to detect any possible mediastinal involvement.

After healing has taken place certain sequelae may have to be dealt with, the most important of which is esophageal stenosis produced by the inflammatory infiltration of its wall and healing by cicatrization.

With a perforation located below the level of the aortic arch, the nature of the surgical procedure will depend upon the time when the patient is first seen. If the injury is recognized immediately and the patient is referred promptly to the surgeon, closure of the opening may be accomplished. The esophagus is exposed through a standard thoracotomy incision, usually on the right side, using an intercostal space varying from the fifth to the seventh depending on

the level of the injury. The closure is made according to the usual principles of technique described elsewhere.

If the injury appears to be so extensive that a resection may be necessary, the left side should be chosen. As a matter of fact, the left side can be used successfully in the majority of instances if the injury is below the aortic arch.

In patients whose perforation has not been recognized or who have been treated conservatively but without success, it may become necessary to drain a mediastinal abscess which has developed in the vicinity of the injury. The technique for this procedure has been described previously (Chapter 14). The inevitable result of the operation, however, is the formation of a fistula to the exterior. This will usually close of itself over the course of several weeks if the opening is small. If the fistula persists, it becomes necessary ultimately to reoperate, at which time in a few instances a closure may be accomplished by mobilizing the esophagus and freshening the edges of the opening. In the majority a resection will be required.

The surgical approach to a *perforation of the abdominal segment* depends once again on the nature of the local condition which varies with the length of time between the accident and the institution of surgical treatment. If the patient is seen early, an abdominothoracic incision should be made in order to gain ready access to the injured segment for closure or for the performance of a resection. The incision should start at the midline, extend transversely across the left upper quadrant and then obliquely backward in the tenth intercostal space through which it is enlarged into the left pleural cavity. The diaphragm is divided partially and an excellent exposure of the lower esophagus is obtained.

If on the other hand it is obvious that nothing but the drainage of a subhepatic abscess can be accomplished, a left upper quadrant laparotomy incision is made. If the abscess is located posteriorly in the subphrenic space, a low lateral transpleural approach provides the best access on the left side. The details of these exposures need not be discussed here. Subsequent treatment for a fistula which fails to close may involve a resection and the abdominothoracic approach may become necessary. This is most unusual.

POSTOPERATIVE CARE follows the usual indications for the hydration and alimentation of the patient and the local management of the thoracic incision and the pleural cavity. Repetition of these details is not required.

Antibiotic medication is continued for a long period or until danger of new infection or spread of pre-existing sepsis has passed.

CHAPTER 24

Foreign Bodies

General Considerations

The gravity of the situation resulting from the lodgement of a foreign body in the esophagus is variable. Left to themselves, even the most harmless and the apparently best tolerated foreign bodies end up by producing complications which are frequently followed by a fatal termination. Any delay in the treatment, any error in diagnosis, or any therapeutic fault often gives rise to a similar ending. The fact is that these disasters can generally be avoided. Although in some instances immediate death may occur, in the great majority the outlook is favorable because of the progress realized in the field of endoscopy during the past two decades.

The penetration of a foreign body into the digestive or respiratory passages is almost always accidental. In rare instances it may be voluntarily provoked either by the person himself, as in hysteria, insanity, or suicidal attempts, or by another person, as in assaults with or without attempts to kill.

The nature of the foreign bodies which may enter the esophagus accidentally is extremely variable. They are usually solid objects, but may be liquids or even gases. The endoscopist will rarely be concerned with liquids except to treat the complications of chemical burns, or with gases which injure chiefly the respiratory tract.

Foreign bodies usually enter the esophagus by way of the mouth, although penetration through a perforating external injury may occur under unusual circumstances (Chapter 22). Their variety is infinite, the only limiting factor being their size which must be small enough for them to be swallowed or otherwise introduced into the lumen. In fact, any object sufficiently small to enter the esophagus may eventually become lodged and constitute a foreign body which must be extracted.

The factors which favor or induce the arrest of a foreign body may have to do with the object itself or with peculiarities of the esophagus. In the first category are the shape, abnormal dimensions, and composition of the object.

FOREIGN BODIES

(irregularities, points, hooks, safety pins, etc) In the second group are the normal or pathological constrictions of the esophagus and alterations of its peristaltic activity

The majority of foreign bodies both in infants and in adults become lodged in the upper third of the esophagus (80 or 90 per cent) Although the question has not been entirely elucidated, this frequency of occurrence in the cervical segment is explained by the physiological narrowing of the organ at the point where it traverses the superior strait of the thorax, as well as by the compression it undergoes from the crowding of other important structures in this region

The vast majority of foreign bodies become lodged as a result of negligence and a great number of these accidents can therefore be avoided Almost all foreign bodies swallowed by infants and young children have been obtained because of thoughtlessness or lack of supervision on the part of those who have them in charge Many children have the habit of carrying to their mouths every object which they can grasp This habit should be combated and no object small enough to enter the digestive tract should be allowed within their reach (but-tons, coins, nuts, beads seeds, coffee beans, etc) Their toys should be large and should not have any part attached which if it became loosened would be small enough for the child to swallow (glass eyes of animals, whistles in animals, etc) Foods should not contain small hard objects which the young child cannot chew into smaller pieces This is especially true before dentition has occurred Vigilant preparation of the food also is necessary to avoid leaving in it any fragments of eggshell, pieces of bone fish bones, bits of enamel from cooking vessels or other extraneous objects which are actually just as dangerous for adults as for children The clothing of the child should not have any brooch or safety pin and for infants and young children tapes instead of buttons should be used as fastening devices

It is also carelessness which causes those who have defective dentures to swallow them while eating or during sleep It is negligence also, this time with a greater responsibility, which leads to the aspiration of surgical or dental instruments or fragments broken off from them, packs tampons, or pieces of tissue such as tonsils or adenoids during their removal

Certain professional habits favor the entry of foreign bodies From the beginning of their apprenticeship all carpenters, upholsterers, dressmakers and cobblers should be trained to avoid using the mouth to hold tacks, nails, and pins

Independently of the factor of negligence so often encountered, additional aspects are sometimes superimposed to aggravate the situation These may even become the dominant factor in making the accident possible Mental disturbances under various forms, from a simple depressed state to complete disorientation with varying degrees of hysteria in between, are frequent predisposing factors Malformations of the air or digestive passages, whether congenital (cleft palate hare lip) or acquired (stenosis compression tumors) either favor the entrance of the foreign body or facilitate its arrest

Disturbances of sensation may be at fault Certain of these are transitory and the risk vanishes when they disappear Syncope, inebriation, anesthesia local inflammatory conditions any of which may alter the sensibility of the

mucous membranes, are typical examples. Sometimes the loss of sensation or hypesthesia depends upon a fixed pathological condition or the remains of a previous disturbance and will be permanent. At other times the alteration of sensation comes from an extrinsic cause, such as the wearing of a dental prosthesis.

Motor disturbances, including paresis or paralysis of the pharynx, larynx, or esophagus, may become the predisposing or causative factor.

In addition there often exist certain adjuvant circumstances, whether accidental or not, which precipitate the accident. These include insufficient mastication, too rapid deglutition (hunger or gluttony), sudden inspiration, cough, straining, falling, fright, sneezing, hiccupping, etc.

Clinical Characteristics

Once the accident has occurred or is suspected, the making of a correct diagnosis assumes great importance. Upon its accuracy and upon the measures which follow depends sometimes the saving of a human life.

Although frequently very definite, the suggestive symptoms must always be looked upon with suspicion because they may be totally imaginary in the insane and in the hysterical or they may be greatly exaggerated in apprehensive subjects. The physician must therefore be on his guard when confronted with a situation which leads him to suspect the existence of a foreign body and must keep in mind the power of suggestion from which the subjects of real accidents of this sort are not entirely free.

The persistence of pain, especially if its location is vague, as well as the persistence of the sensation of a foreign body being present, are symptoms too suggestive not to have a probable value. However doubtful the reality of the presence of a foreign body in the patient may be, the physician should never abandon the idea without having made certain that the accident is imaginary, and he must not dispense with various examinations, even if eventually an esophagoscopy becomes necessary, to arrive at such a conclusion.

The signs are often clear and their objectivity makes the reality of the accident immediately obvious. In such a case the various investigations carried out serve only to confirm the fact. In other instances the evidence is nonexistent or has not been appreciated, especially if the accident involves a young child in the absence of witnesses or an adolescent or adult who is unconscious as in sleep, syncope, drunkenness, anesthesia, or coma, or who is mentally irresponsible. Sometimes also, the lodgement of the object has actually been silent or the symptoms so slight that even a careful observer might not have detected them. When this occurs, it is only the pathological secondary manifestations which give rise to suspicion. The diagnosis in these cases becomes more difficult and the examinations more intricate.

The variable symptomatology depends upon a series of factors of varying importance which gives to each case a characteristic of its own. Foreign bodies, in fact, are never the same, and although there are certain general rules concerning the etiology, the diagnosis and the treatment, each foreign body presents

certain particulars which simplify or complicate the diagnostic and therapeutic problems which it imposes

Symptoms

The *symptomatology of foreign bodies lodged in the esophagus* comprises two types of manifestation which are sometimes indistinguishable but which are frequently distinct and different. First are those of the period of invasion or acquisition of the object, and second, those of its continued lodgement. Independently of the chronological order of their appearance, the symptoms can be subdivided into (1) those of obstruction, (2) those of irritation, and (3) those of inflammation.

The FIRST GROUP of symptoms is generally caused by the foreign body itself and follows either the ingestion of the object or its dislodgement. As opposed to these obstructive phenomena which may be called *primary*, there arise *secondary* manifestations of obstruction which come later and are caused by inflammatory reactions engendered by the presence of the foreign body itself.

The symptoms belonging to the SECOND GROUP may also be immediate although they are usually secondary. They may be extremely variable according to the nature and shape of the foreign body, the duration of its sojourn in the patient and the reactive phenomena to which it may have given rise.

The symptoms of the THIRD GROUP are never immediate although in certain instances they may develop early. The factor of infection becomes predominant and the local effects often become eclipsed by the complications. Here also the characteristics of the foreign body itself and the duration of its presence become as important as the powers of reaction of the patient or his eventual lack of resistance.

SYMPTOMS AT THE TIME OF LODGEMENT *Embarrassment of deglutition* is frequent but transitory and of variable intensity. If prolonged it eventually leads to dysphagia.

Coughing though unusual is sometimes experienced. This symptom may lead to the erroneous conclusion that the patient has inhaled the foreign body and that it lies in the larynx, trachea or bronchi. The cough is usually convulsive and subsides rapidly.

Regurgitation, which must be distinguished from vomiting with which it is often confused, is very frequent when the foreign body is swallowed during the course of a meal. Sometimes it serves to expel the intrusive object soon after ingestion. The liquid which is regurgitated may consist only of esophageal secretions. It may be blood-tinged or frankly bloody if the foreign body is sharp and traumatic.

Pain is a rather constant symptom. It may be moderate or severe. It may be transitory, when it will diminish in intensity or rapidly disappear, or it may persist as long as the object remains impacted in the esophagus. Occasionally there is no pain.

The pain is caused by sharp points on the foreign body, local injury created by the lodgement of the object, the distention provoked by obstruction by a large object and the esophagospasm secondarily produced, particularly at the orifices of the esophagus. The location of the pain is variable. It may be high

in the epigastrium, subxiphoid, retrosternal, or in the back. It may not correspond exactly to the zone of impaction of the object. It is for this reason that it is often a rather unreliable symptom, especially since it may persist after the spontaneous expulsion or the instrumental extraction of the offending object. Occasionally it is elicited only by deglutition or palpation over the region where the impaction has occurred.

A subjective sensation of the presence of a foreign body which is in fact only a modified form of pain or discomfort is a more reliable symptom than pain itself. But it may be merely imaginary, as in an hysterical subject, or represent an erroneous interpretation on the part of the patient of disturbances of sensation in certain neurological disorders. In other instances it may be an expression of the persistence of sensation provoked by the passage but not the arrest of a large foreign body. It is a symptom, however, which has real value in subjects who are rational and objective.

Dysphagia, the most frequent and the most constant symptom, occurs immediately and usually becomes progressive. It may reach any degree of importance from slight intermittent blockage, occurring only while swallowing solids, to complete and permanent aphagia with inability to swallow anything. It is usually painful. In the occasional patient who has no pain even in the presence of large objects like coins or dentures, it is the expression of a mechanical obstacle to the ultimate passage of the food. The dysphagia varies depending in general upon the volume of the foreign body, the degree of obstruction, the amount of inflammation, and the intensity of the associated esophagospasm.

SIGNS OF THE PERSISTENCE OF THE FOREIGN BODY are usually a continuation of those experienced when it first becomes lodged. Cough is exceptional but it may appear or persist because of nerve reflex activity or because of the aspiration into the larynx of saliva or esophageal secretions.

Regurgitation of food, often aggravated by actual vomiting, is much more frequent and tends to grow progressively worse. The same is true of the pain and dysphagia. In fact, the initial symptoms evoked by the lodgement of the object are aggravated by the effects of obstruction and inflammation which the presence of the foreign body provokes. These reactions are in general dependent upon the duration of the period of impaction and induce an increase in the severity of the symptoms.

Fever, which tends to increase in degree if the foreign body is allowed to remain, provides evidence of the onset of inflammation due to infection. It is a warning sign signifying the urgent need to extract the offending object. The cause of the fever should be sought because it is often extra-esophageal in origin. Pulmonary complications, abscesses, and empyema are the most frequent conditions encountered. This is important in deciding what treatment other than the extraction of the foreign body may be indicated.

Hematemesis, although unusual, is a disturbing sign, for it indicates the presence of an injury or an actual perforation of the esophageal wall. The appearance of this sign frequently makes the prognosis more serious because of the probability of complications to which it may lead.

There is also a group of laryngeal or bronchopulmonary signs which may confuse the diagnosis. They are due to spill over into the larynx and aspiration

of esophageal secretions, mucus, liquids swallowed, etc., into the tracheobronchial tree

Sometimes an esophagotracheal fistula, an injury to the larynx induced by digital or instrumental maneuvers, or a lesion of the recurrent nerve may be the cause. The differentiation from foreign bodies in the air passages may be made very difficult.

Signs

The PHYSICAL EXAMINATION, both external and internal, must be methodical. The pharynx and hypopharynx should be scrutinized with care. A superficial erosion, an abnormal inflammatory condition, or edema behind the larynx is evidence in favor of the presence of an esophageal foreign body. The abnormal presence or stagnation of secretions in the pyriform sinuses or in the hypopharynx should be looked for (Chevalier Jackson sign). Although this finding is not pathognomonic and although it denotes merely the presence of an obstacle to the free passage of secretions down the esophagus, it is the most reliable objective evidence of foreign bodies in this organ. Given appropriate additional evidence it aids materially in the diagnosis of an esophageal foreign body.

Percussion and auscultation are of little value in the diagnosis. They may give evidence of pulmonary complications but do not provide information concerning the condition of the esophagus or the existence of a foreign body.

Palpation of the cervical esophagus, which is the most frequent location of a lodged foreign body, will disclose tenderness coinciding in general with the place where the object is caught. The localization of pain and tenderness is sometimes very exact and palpation assumes a great importance in these patients.

Roentgen Examination

The roentgen examination is of inestimable value and must never be omitted. From the radiological viewpoint, foreign bodies should be grouped into two categories: those which are radiopaque and those which transmit the roentgen rays.

In the former group there are all degrees of opacity depending on the nature of the object, but it is useful to divide them into those which are strongly radiopaque, like metallic objects, and those which are slightly radiopaque, like thin metallic bodies or fragments of bone.

Foreign bodies which are STRONGLY RADIOPAQUE make it possible to establish the diagnosis at once. Their shadow can be seen under the fluoroscope even if it is outlined over that of the vertebral column, the ribs, the heart, or even a combination of these shadows (Fig. 303). With these it is necessary merely to take note of the contours of the object by examination in different positions and to take films in two planes at a 90 degree angle from each other. As a general rule, one film is made in the anteroposterior and another in the lateral axis. Certain peculiarities of the object may be visible only in a lateral or an oblique view, in which case a film is made in the appropriate position (Figs. 304 and 305).

With objects which are ONLY SLIGHTLY RADIOPAQUE the examination must



FIGURE 303 Roentgen film of radiopaque foreign body in the esophagus, a complicated case with part of the object in the esophagus and part in the stomach. Solid objects of this type cast a shadow which is easily differentiated from all the superimposed visceral and skeletal shadows (vertebral column, heart, liver, stomach, etc.) (Meyers)



FIGURE 304 Re-
old. The fact that
film (A) In the upper
there is one coin over

showing two coins
jects together is no
shadow in the fr
(Chevalier

her. In a child 2½ years
except

Cl

be very minute from zone to zone and in various positions along the entire length of the esophagus. This may disclose a suspicious shadow visible in only one particular position. A film made in that position often makes it possible to establish the diagnosis (Fig. 306). The discovery of a weakly radiopaque foreign body rarely gives precise information regarding its nature or its exact shape.

It is useful to recall that every esophageal foreign body, if it is more or less flattened in shape, always becomes lodged in the frontal plane. It is therefore in this axis that the opacity of the object is maximal and that the examination can best disclose it.

In the group comprising the foreign bodies which are **RADIO-*LU*CENT**, fluoroscopy may be the only objective means of determining both the presence and the position of the object and thus is of major importance. These facts may be elicited only by a study of the swallowing function with the purpose of disclosing any abnormalities of its mechanism. This investigation should be carried out with the assistance of a radiopaque medium. In cases giving a negative result the ingestion of a capsule containing bismuth or Lipiodol should be tried. Various situations may be encountered:

1 *The foreign body may be causing obstruction.* The barium column in these patients stops when it reaches the obstacle and accumulates above it, giving in silhouette the shape of the upper extremity of the object. Thus one may establish the location of the obstacle, the size and shape of the foreign body, and sometimes its nature (Fig. 307).

2 *The foreign body may be only partially obstructing the esophagus.* In these the column of opaque material stops momentarily and then filters more or less rapidly along or through the foreign body. Often the column may be seen to double back upon the obstacle. In whatever way the foreign body may be irreg-



FIGURE 305 Foreign body seen in the oblique projection only. Rabbit bone in the cervical esophagus. (Courtesy of Prof. DeBacker.)



FIGURE 306 Roentgenogram of a foreign body difficult to visualize. Chicken bone not seen in the frontal view because of laryngeal and vertebral shadows clearly visible in the lateral view. This object was actually invisible under the fluoroscope and was seen only after the lateral view film was developed (Courtesy of Prof. DeBacker)

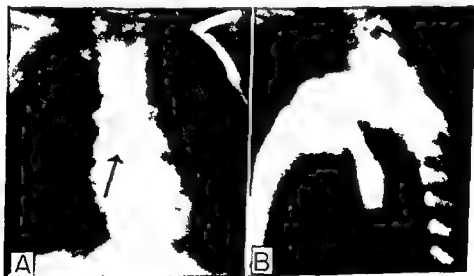


FIGURE 307 Roentgenograms of a nonopaque obstructing foreign body in the esophagus. The object is a nut lodged in the thoracic segment. The barium mixture outlines the superior surface of the object (Chevalier Jackson Bronchoscopic Clinic)



FIGURE 308 Roentgen film after ingestion of barium. Nonopaque foreign body consisting of a wooden comb revealed by the presence of the barium (E. Huizinga)

ular in shape or present sharp angulations bits of barium mixture will adhere to its surface and delimit its contours (Fig. 308)

3 *The foreign body may not be obstructing* In this instance the barium column passes into the stomach without delay. The radiopaque capsule, used as a last resort in diagnosis, becomes extremely valuable in this situation because it will stop or at least hesitate a moment before resuming its course after interruption by the foreign body (Fig. 309)

Even when the various methods of examination just described are negative, the roentgenologist is not in a position to conclude that no foreign body is present

Esophagoscopy

If the slightest doubt exists or if a suspicious symptom appears ■ diagnostic esophagoscopy must be performed. When carried out by ■ skilled endoscopist, this examination ■ painless and absolutely innocuous. It permits visualization



FIGURE 309 Roentgen films showing the use of a capsule filled with barium to indicate the location of an impacted nonopaque foreign body in the esophagus. Fragment of bone in the cervical segment of the esophagus. The foreign body is invisible in both the frontal (left) and the lateral view but the arrest of the opaque capsule demonstrates the existence of an obstacle (Dr Manges)

of the lumen of the esophagus throughout its entire extent and removes any lingering doubts. If the examination discloses a foreign body which previous methods of investigation have not revealed, the extraction of the object should be undertaken at the same sitting provided that all the precautions necessary have been taken.

Differential Diagnosis

The establishment of a correct diagnosis of impacted foreign bodies demands special consideration on the part of the physician.

In spite of the extraordinary diversity of the direct manifestations provoked by the lodgement of the object in the esophagus, certain general principles applicable to all cases should never be lost sight of. These are as follows:

1 The physician should never allow himself to be influenced by the agitation of the patient or of his entourage. It is important for him to remain calm and, above all, to impose the same attitude upon others who may be present.

2 It is wise, whenever suspicion is aroused, never to deny from the first the possibility that a foreign body may be present. In addition to the fact that the prestige of the physician may suffer if his assumption that a foreign body was not present proves to be erroneous, the prognosis is likely to be unfavorably affected if treatment is delayed. The existence of a foreign body must be admitted after the various examinations are in agreement that one is probably present.

3 One must listen to the patient and those who are with him. A careful

history should be obtained by asking clear and precise questions, the affirmative or negative responses to which serve to assist in the making of the diagnosis

4 Having in mind the multiplicity of symptoms which a foreign body may cause, one should be ready to alter a first impression that may perhaps be erroneous. One should remain objective and avoid preconceived ideas

5 All impetuous manipulations, either on the part of the physician or of anyone else, must be avoided

6 One should remember that the situation is seldom so urgent as to make it proper to dispense with a careful history and an examination as complete as possible, as well as detailed preparation for exploratory intervention or an effort to extract the object

Interrogation about the history should be conducted with attention to detail from the patient as well as from the parents or other witnesses. The symptoms of the onset should be sought such as choking, respiratory spasm, cough, cyanosis, asphyxia, pain, obstruction, regurgitation, vomiting, or dysphagia. The presence of one or several of these symptoms is not pathognomonic but warrants the presumption that such an accident may have occurred. Further developments such as recovery after the initial attack, persistence of the symptoms, aggravation of symptoms, or appearance of new ones should be observed.

The neck, chest, and abdomen should be examined and any abnormalities such as the following noted: bending and immobilization of the head, swelling of the neck, inspiratory retraction of the lower chest, and partial splinting of the chest. Percussion and auscultation may reveal suggestive pulmonary signs or even actual evidence of abnormality. The nose, mouth, oral cavity, pharynx, larynx, and hypopharynx should be inspected.

The results of the physical examination may be conclusive, doubtful, or negative, but they are usually completed by a *roentgenological investigation*. This may not always be obligatory, but it is frequently necessary and almost always useful. Both fluoroscopy and the taking of films are employed. The examination must conform to certain elementary rules of observation upon which its efficacy depends. These are:

1 One must never be satisfied with only a quick glance at the fluoroscopic screen. The fluoroscopic examination must be complete and never superficial.

2 As with every roentgenological investigation, the eyes of the examiner must be well accommodated. The time for this to take place varies with the individual and the intensity of the illumination outside, but at least ten minutes' time is required for dark adaptation.

3 In addition to the fluoroscopy, various films should be made to permit study of the contours and location of a given object. Frequently in spite of a negative fluoroscopy, the roentgen films disclose an opaque foreign body of small size or a larger object of somewhat lessened opacity, which the eye may not have been able to recognize on the fluoroscopic screen (Fig. 306).

4 Films should be made in several positions because flat objects or those with a reduced opacity are often discernible only in a single projection (Fig. 305). The same is true of certain foreign bodies, the multiplicity of which is discovered only by this means (Fig. 304). Views in different projections are equally valuable for the study of the contours of the object.

In case of failure of the above-mentioned diagnostic procedures, if there is the least suspicion that a foreign body is present, direct endoscopic visualization should be carried out. A careful check of the interior of the organ will almost certainly resolve the problem. The efficacy of this method is great and, if positive, this simple examination may become the occasion for the extraction of the impacted object. In expert hands the inconveniences of the procedure are entirely negligible when compared with the definite risk which the presence of an undiscovered foreign body presents.

Prognosis

Improvements in operative techniques, hand in hand with the remarkable perfection of endoscopic instruments during the last thirty years, have completely transformed the prognosis. Formerly considered very grave, since the mortality was as high as 58 per cent, the prognosis of foreign bodies was notably improved by the beginning of this century when the mortality fell to 8 or 9 per cent. The recent progress of endoscopy, to which great impetus was given by the American school, thanks to Chevalier Jackson, has transformed this prognosis, formerly so serious, to one which is actually favorable. The impressive statistics of Jackson and his disciples reveal a mortality of less than 2 per cent and, since 99 per cent of the total number of foreign bodies can be extracted from the esophagus, the percentage of clinical cures surpasses 98 per cent.

These remarkable results are no longer exceptional. Although the nature of the foreign body, its location, the duration of its existence, and the age of the patient are important factors which influence the prognosis unfavorably or, on the contrary, make it better, the factor of first importance is independent of the foreign body itself or even of the patient. The prognosis, in fact, depends chiefly on the practical knowledge of the endoscopist and even more upon his surgical skill.

If foreign bodies are left to themselves, however, the outlook is bad because of complications, either early or delayed, which frequently end in death. These complications are acute esophagitis, esophageal abscess, perforation of the wall of the esophagus, mediastinitis, esophagotracheal fistula, empyema, pulmonary sepsis, acute dehydration, and hemorrhage from perforation of the aorta. Cervical spondylo-arthritis, sometimes with compression of the spinal cord, should also be mentioned. As a late complication a stricture may be encountered.

CHAPTER 25

Extraction of Foreign Bodies from the Esophagus

Introduction

Given the presence of an impacted foreign body, one of two diametrically opposed courses might be adopted—passive purely expectant treatment or active intervention. Unless the object has passed on into the stomach, from which spontaneous expulsion after a delay of two to five days is the rule, a passive attitude is undefensible. Denoting an exaggerated optimism, it fails to take into account the rarity of peroral expulsion of objects impacted in the esophagus (not over 2 per cent). By prolonging the duration of their stay in the esophagus, a course of inaction favors the development of complications and renders the ultimate extraction of the object more perilous and more difficult.

An active approach to the problem therefore is the proper course, but it must be thoughtfully planned and prudently carried out. Chevalier Jackson has adopted the principle that every foreign body which has entered by the natural route should be extracted by the same route. In spite of its apparently intransigent absolutism this principle will be found to apply in all but the most exceptional cases.

Once it is decided that the object can be extracted, the most efficacious and least traumatic technique which can be expected to succeed must be adopted.

Blind attempts to push a foreign body lodged in the esophagus into the stomach by means of a bougie or sound as well as the use of a DeGraefe basket should be mentioned to be definitely condemned. The Kirmisson hook still has several advocates. If manipulated with care under the fluoroscopic screen this instrument may succeed in extracting obviously opaque objects from the upper or middle portions of the esophagus provided they do not have sharp irregularities. The extraction of coins or similar objects is the only justifiable

use of this method. The same remark and the same limitations apply to the use of a curved esophageal forceps.

The last two instruments may permit an adroit operator to relieve his patients without serious risk when it is difficult to obtain the service of an experienced endoscopist. Their use, however, should be strictly limited.

External esophagotomy is also a procedure which has a limited but definite use. Applicable usually to foreign bodies lodged in the upper third of the esophagus, this method should be attempted only when peroral extraction presents insurmountable difficulties and when an endoscopic attempt made by an expert has failed. In exceptional cases a foreign body may have to be extracted by esophagotomy from the thoracic portion of the organ through a thoracic incision.

Extraction under esophagoscopic control constitutes the method of choice. It is adaptable to almost all cases, it permits the resolution of almost all the mechanical problems posed by foreign bodies impacted in the esophagus, and offers guarantees of absolute security in the hands of a well trained operator.

The one exception to this rule is large particles of food which may ultimately become softened and pass on. In this instance if the impacted bolus is a large fragment of meat, as it so often is, a solution of 5 per cent papain in water swallowed in doses of one teaspoonful every half hour will invariably dislodge it in a few hours as the proteolytic action of the papain takes effect.

Otherwise active intervention must be initiated unless there is a serious contraindication. Even the presence of an aortic aneurysm or a serious cardiovascular disorder, or the extreme youth or advanced age of the patient does not constitute a contraindication. They merely stimulate the operator to employ more than the usual caution.

Technique

General Principles

Endoscopic extraction is to be practiced according to the rules of technique regarding the introduction and manipulation of the esophagoscope. The mobilization, grasping, and extraction of the foreign body comprise problems which are purely mechanical. These problems, which vary depending on the characteristics of the foreign body and of the tissues which surround it, must be studied and solved in each individual case. The application of certain general rules, however, is essential to the success of the extraction in all. Although they may seem to be self-evident, these rules are too frequently disregarded by the occasional endoscopist and they should therefore be emphasized. Their omission is the most frequent cause of failures which can scarcely be condoned. These rules are as follows:

- 1 One must see what is being done. If it is impossible to see, it is absolutely essential to know what is taking place beyond the field of vision.
- 2 The foreign body must be exposed. This means that after its location is established, the ideal exposure for its extraction should be sought. If this is not possible as it lies, the object should be moved into correct position by manipulation with instruments or by pressure upon the foreign body itself (in the neck).

3 Before any attempt is made to grasp the object, one must be able to see or create a *free space* for the application of the jaws of the forceps

4 A forceps must be chosen which is adapted to the role which it must fulfil in respect to the dimensions the shape and the consistency of the object

5 The grasping of the object by the forceps must be *correctly executed* Halfway measures will not do Above all, the hold on the foreign body must be *secure*

6 During the extraction one must not attempt to overcome any resistance which may be encountered The extraction of a foreign body is a maneuver which must be *carried out gently*, not by the application of force

7 The tissues of the esophagus must be protected against any possibility of trauma from sharp points or jutting edges of the object which might lead to an injury or a perforation

8 In the vast majority of instances the foreign body must be held in contact with the end of the esophagoscope throughout the duration of its removal

Anesthesia

The indications for general anesthesia are extremely rare It should be employed only in exceptional instances, as when the cooperation of the patient cannot be obtained

Local anesthesia of the pharynx and hypopharynx obtained by the application of a 5 to 10 per cent cocaine solution or other topical agent in appropriate concentrations is sufficient to permit an easy and painless esophagoscopy The acuity of the reflexes as well as the anxiety of the patient may be diminished by the hypodermic administration of a hypnotic agent such as a mixture of morphine and scopolamine one hour before the intervention Although very useful this preparation of the patient is not indispensable and may be omitted in the rare instances in which haste is important With infants and young children, any form of anesthesia is usually either useless or contraindicated

Extraction of Soft Foreign Bodies

These are usually particles of food Meat is the most frequent offender, either because a piece is too large to pass through a normal esophagus or if smaller in size, because it becomes lodged at a pathological obstacle such as a stenosis a tumor, an angulation or a kink The obstruction is often complete with rapid accumulation of secretions above the object which makes preliminary aspiration essential

The object should be grasped with a forceps having jaws with a large surface for contact A flat forceps or, better still one with fenestrated jaws of large size should be used In this way the morcellation or breaking up of the foreign body which prolongs the procedure may frequently be avoided If morcellation should occur in spite of this the fragments can generally be extracted one by one through the lumen of the esophagoscope without removing the tube The fragmentation of a soft foreign body is more bothersome than tragic The ultimate success of the procedure is not compromised and the chances of cure are not diminished

If the object is sufficiently compact and the grasp upon it sufficiently secure

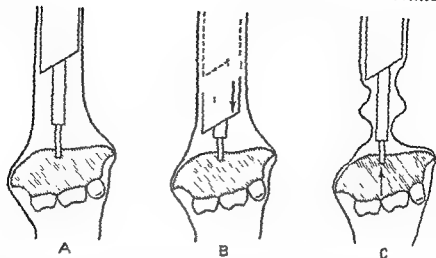


FIGURE 310 Extraction of a denture showing the principles involved in avoiding injury to the esophagus. *A*, Grasping of the foreign body. *B*, Correct start of extraction, the esophagoscope is brought into proximity with the foreign body before any traction is exerted on it. The esophagoscope thereby serves to push aside the esophageal walls during extraction. *C*, Faulty attempt at extraction, traction exerted on the foreign body by means of the forceps produces wrinkling of the esophageal wall and the interposition of ridges of wall between the foreign body and the end of the esophagoscope. The object becomes stuck and easy extraction becomes impossible. Forceful extraction under the circumstances is extremely traumatic and probably lethal.

so that it can be freed and lifted out in one piece, the esophagoscope should be kept in contact with it as it is being withdrawn, the instrument thereby preparing the way for the object to follow. If the grasp is correct, the tube is pushed gently as far as the foreign body, before proceeding with the actual extraction. The foreign body must not be drawn up to meet the tube. This rule is subject to rare exceptions, as when the object must preserve a certain degree of movability between the jaws of the forceps in order to permit it to pass around obstacles which may be in the way. In withdrawing the instrument along with the foreign body, the walls of the esophagus are parted. On the other hand, if the foreign body is drawn up to the instrument while the latter is held immobile, it often pulls or drags along some folds of mucosa which get in the way between the object and the end of the tube. This may make extraction impossible (Fig. 310).

Extraction of Hard Foreign Bodies

The problem of grasping firm objects is very different from that with soft ones. If correctly applied, the grasp of the instrument should hold and the foreign body will not break into pieces during the manipulations of extraction. The esophagus, however, must yield and being less resistant may suffer damage from the effect of immoderate traction.

An injury to the wall of the esophagus or pharynx or a perforation is very serious. Hard foreign bodies alone may cause it. Perfection of the technique of extraction therefore, assumes pre eminent importance in such cases. With certain foreign bodies, notably those which are pointed, the slightest technical error may result in a fatal outcome.

Some foreign bodies like blown glass beads are hard but fragile, and the

correctness of the hold upon them must be attended by the greatest delicacy of the grasp

Hard Foreign Bodies of Regular Shape

Although the characteristics of their surfaces may render their prehension difficult, this same quality eliminates or at least diminishes the risk of lacerating the walls of the esophagus. The forceps will lose hold of the object under a slight effort of traction before any damage to the organ occurs. The surgical risk is thereby reduced. The problems presented by various types are as follows:

FLAT OBJECTS The largest number of objects in this category are coins, the frequency of which is particularly great in children. Buttons, metallic whistles, and similar objects present the same characteristics as much from the mechanical as from the pathological point of view.

For anatomical reasons a flat foreign body in the esophagus always presents in the transverse plane although the same object if it becomes lodged in the trachea or a bronchus will lie in the anteroposterior plane or in an oblique position. In addition to every other clinical sign, this single peculiarity of flat foreign bodies makes possible an immediate differential diagnosis between their presence in the esophagus and in the trachea.

Furthermore a flat object rarely occludes the lumen of the esophagus. It may distend it, but the organ retains its relative patency. The pressure of such an object may eventually cause an inflammatory swelling which eliminates this patency or permits the accumulation above it of particles of food which add to the obstruction.

A flat foreign body usually sticks to the wall of the esophagus by one of its surfaces. Edema of the mucosa or of the rugal folds of the wall may then partially obscure it. This is exemplified by the difficulty experienced in the discovery of a coin, the exact location of which is known. Most often the piece lies flat against the posterior wall of the esophagus where it is concealed from



FIGURE 311 Foreign body at the mouth of the esophagus concealed by the bulge produced by the cricopharyngeus muscle beneath the esophageal mucosa. A Cricopharyngeus muscle. B Cricoid cartilage (Coin lying distal to the cricopharyngeus)



FIGURE 312 Foreign body below the mouth of the esophagus concealed by a fold of esophageal mucosa pushed up by the pressure of the end of the esophagoscope during its introduction.

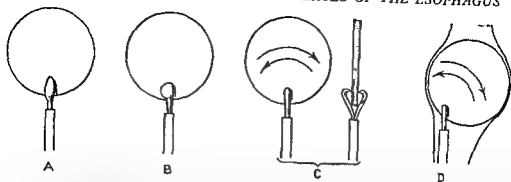


FIGURE 313 Extraction of a flat foreign body. Correct grasp of a flat foreign body like a coin. The grasp is made near the edge of the object by means of a straight forceps (A) or a forceps made for lateral grasping as at B. Better still is a rotation forceps as at C. The latter insures great movability of the foreign body and at the same time effects a secure grasp on the object (D).



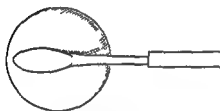
FIGURE 314 Extraction of a foreign body which has a rim around the periphery like a button. The bite of the rotation forceps is very secure, thanks to the raised rim around the edge of the object.



FIGURE 315 Extraction of an oval foreign body (incorrect technique). The oval shape of the object causes the forceps to slip when applied to the edge.



FIGURE 316 Extraction of an ovoid foreign body (correct technique). The spoon-shaped jaws applied to the meridian of the object do not permit the escape of the foreign body from their grasp.



the operator's view either by the posterior fold made up of the cricopharyngeal muscle or by a slight fold of mucosa produced by advancing the esophagoscope (Figs 311 and 312).

The foreign body may also be passed over by the esophagoscope without being seen, but the contact between it and the metallic tube will usually be recognized. It is often during the withdrawal of the instrument, while searching the esophageal wall from below upward, that the foreign body is finally discovered. This is likely to be only a fleeting glimpse, however, because the movability of the esophageal wall augmented by the respiratory motions, by coughing, and by the movements of the patient often obscures it. The operator should therefore be prepared to seize the object on the fly while the presentation is favorable and a correct grasp is possible.

The bite of a straight forceps with flat jaws, or better of a rotation forceps, should be applied on the edge of the coin. The rotation forceps, which permits a very firm grasp, has the advantage of allowing the coin to pivot or swing upon

FIGURE 317 Extraction of a foreign body which has a rough surface. The points of a rotation forceps find a solid grip in the irregularities of the surface

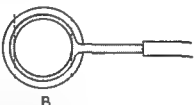
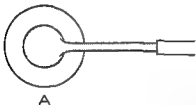
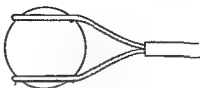


FIGURE 318 Extraction of a spherical foreign body using a forceps which has either cup shaped jaws (A) or annular jaws (B)

the axis of the grasp and to move easily around the obstacles which it encounters while the esophagoscope forceps and foreign body are being withdrawn together (Fig 313)

Some flat foreign bodies such as buttons have a slightly raised thickened edge which makes them easy to grasp (Fig 314). Others may have a sloping edge and a firm smooth surface on which every forceps tends to slip (Fig 315). In this situation the only possibility of extracting the object is to use a straight forceps with long slightly curved jaws which grasps the rounded object at or beyond its center (Fig 316).

SPHERICAL OBJECTS. This group of foreign bodies includes beads, pearls, pits, nuts and other objects of similar shape. If the surface of such an object is rough, as for example that of a peach pit, the rotation forceps can be employed and the grasp should be good even if the bite is not applied at the equator of the object (Fig 317). If the surface is smooth and at the same time hard, the only possibility is to apply a forceps with cupped or curved jaws beyond the equator (Fig 318, A). Beads and other spherical objects must be grasped with a forceps having ring-shaped jaws (Fig 318, B).

Hard Foreign Bodies of Irregular Shape

The variety of these objects is infinite and no precise classification can be given. The endoscopist may group them according to the difficulties and the risk which their removal entails as (1) those with nontraumatizing irregularities, (2) those with traumatic sharp angles or edges and, as a special group of the latter, (3) those with points which might perforate.

1 So far as irregular foreign bodies with *nontraumatizing angles* are concerned, each one constitutes a particular problem which must be solved according to the rules of procedure already elucidated. If at all possible, before extrac-

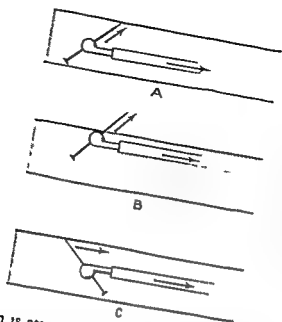


FIGURE 319 Extraction of a pin. The problem of the point. Any traction exerted in the direction of the point causes the point to penetrate the soft tissues against which it lies (A and B). If the point drags the tissues may at the most be scratched without serious risk and without perforation (C).

tion is attempted in a patient, the proposed solution of the technical problem should be tried on a rubber model or in a dog using an identical or similar object. This preliminary study permits one to modify the technique originally decided upon if it proves to be faulty. More important still, it provides the operator with a dexterity which insures success, whereas an attempt made without careful study or one entirely improvised may be doomed to failure.

2 The same comments and the same manner of proceeding apply to foreign bodies which have *traumatizing angles*. The necessity for a perfect technique here is still greater, for to the risk of failure is added the more serious risk of causing injuries to the esophagus which the operator should have been able to avoid and which might lead to the death of the patient.

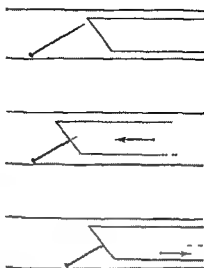
The sharp edges of the foreign body must be covered or protected either by the esophagoscope or by the forceps. It is always better to fail and preserve the integrity of the tissues than to succeed in extracting a foreign body at the price of producing an injury. The first eventuality permits subsequent attempts and leaves room for an ultimately favorable outcome, whereas the second is often fatal for the patient.

3 Foreign bodies with *perforating points* are by nature less varied. Their extraction comprises certain precise rules from which there must be no departure. This type of foreign body includes certain bones, particularly fish bone and all pointed objects like nails, needles, various types of pins, staples, etc. In the extraction of these it should always be kept in mind that every object which moves with its point forward is likely to penetrate wound, and eventually perforate. This same point if it trails behind is completely innocuous (Fig. 319).

If the foreign body has entered the esophagus point forward, which is unusual, the mechanical problem is simple for the point will trail behind during the extraction and no important injury need be anticipated. When the point is directed proximally, which is the usual situation, the problem assumes great importance. The risk increases and the technique of extraction must be perfect.

If there is only one sharp point, the operator must look for it. It may be free and easy to locate. The esophagoscope should then be advanced carefully.

FIGURE 320 Extraction of a pin. The free point is engaged in the end of the esophagoscope before it is grasped. The pin may then be extracted without risk either by way of the esophagoscopic lumen or along with the withdrawal of the entire instrument.



until the point has entered the lumen of the instrument. It is only after this result has been obtained and the esophageal mucosa is thereby protected by the walls of the tube that the pointed extremity of the foreign body should be grasped (Fig. 320).

Although it may be free, the point is often stuck against the wall of the esophagus. In this instance the end of the esophagoscope may be used as a lever to push away the mucosa in order to engage the point (Fig. 321).

More often the point will have penetrated more or less deeply into the tissues. It must then be disengaged and drawn into the lumen of the esophagoscope. This disengagement is accomplished by pushing the foreign body downward with a forceps. The point then slides out of the tissues and is finally recovered by the esophagoscope. This maneuver is accomplished best by insinuating one of the jaws of the grasping forceps between the foreign body and the mucosa as close as possible to the spot where the object has penetrated. A slow, down

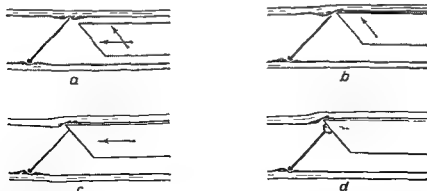


FIGURE 321 Extraction of a pin. When the point is engaged in the mucosa (a) it must first be freed by lateral pressure with the end of the esophagoscope against the wall of the esophagus (b) before being engaged by the end of the tube (c). It is only after the execution of this maneuver that the pin should be grasped and then the point must be seized as it presents in the end of the instrument (d).

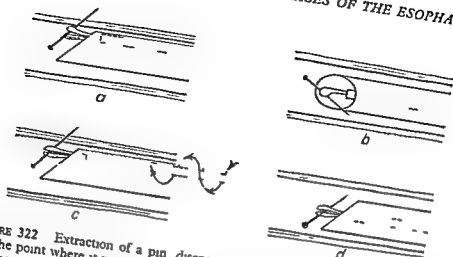


FIGURE 322 Extraction of a pin. *a* The pin is grasped close to the point where it penetrates the esophageal wall. *b* Same as *a* viewed from above. *c*, Disengagement by a movement of rotation and simultaneous propulsion exerted by the forceps. *d*, The entire pin is now in position to be drawn into the lumen of the esophagoscope.

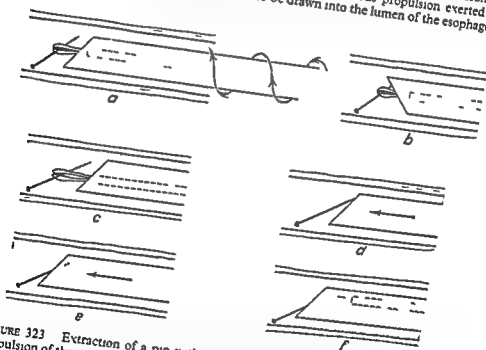


FIGURE 323 Extraction of a pin with protection of the point. *a*, *b* by simple rotation and propulsion of the esophagoscope. The end of which ultimately covers the pointed extremity of the pin. *c*, *d*, *e*, *f* by manipulation and final grasping of the point of the foreign body in the interior of the tube.

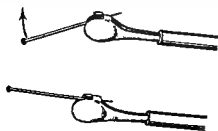


FIGURE 324 Extraction of a pin using the Tucker forceps. Thanks to the hook with which the edge of each of the spoon shaped jaws is provided the forceps has a tendency to direct the foreign body into the axis of its stem.

ward, twisting movement almost always succeeds in directing the point into the esophageal lumen (Fig 322)

The shielding of the point is then accomplished either by simple rotation of the esophagoscope or by advancement of the tube with or without twisting (Fig 323, *a, b*) This maneuver often demands the withdrawal of the forceps and a new grasp applied this time on the extremity of the foreign body whereby the point is entirely protected (Fig 323, *c, d, e, f*) The use of the Tucker forceps facilitates the realignment of the point in the axis of the instrument (Fig 324)

The general rules enunciated above apply to all pointed foreign bodies including safety pins, nails, and ordinary pins

Special Problems

Various artifices permit the resolution of certain problems One example is the bending of the pointed object if it is made of MALLEABLE MATERIAL (Fig 325) Another is cutting the object which is to be avoided if possible (Fig 326)

STAPLES The point may be double, as in the case of a staple One or both of these points may lie buried in the wall of the esophagus The principle of disengagement of the point and of its protection must be observed in this instance, but the application is not easy The two points may be protected either by the tube or by the combination of the tube and the forceps or by the use of a special forceps (Fig 327) The most commendable method however is to turn the staple around within the esophagus if the respective dimensions of organ and object permit The cephalic version of a double-pointed foreign body like a staple may be accomplished in the plane of the arms of the object or in the axis of its points (Fig 328)

SAFETY PINS The extraction of safety pins often presents serious difficulties and grave risks Chevalier Jackson describes sixteen different methods,

FIGURE 325 Extraction of a pin (bending technique) This is carried out by means of a special forceps the ends of which can be withdrawn into the interior of the large strong outer sheath The method can be used only with flexible or malleable objects

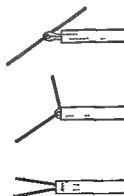


FIGURE 326 Extraction of a pin Method of cutting the pin (a technique which is ill advised)



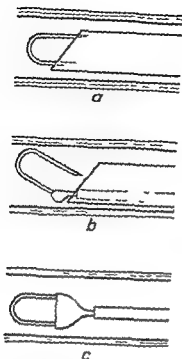


FIGURE 327 Extraction of a staple
Protection of the points of a staple by
the esophagoscope only (a) the esophago-
scope and a forceps (b) and by any of the
various models of special forceps (c)

FIGURE 328 Extraction of a staple
Version of a staple in the plane of its
branches (A) or on the axis of its points
as at B (After Chevalier Jackson)

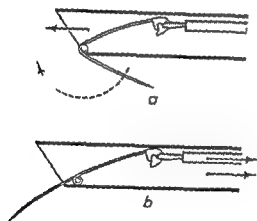


FIGURE 329 Extraction of a safety
pin Method of straightening the pin
with the hood held immobile in the in-
terior of the esophagoscope by means of
a forceps the tube is pushed gently and
firmly downward Resting on the pointed
arm of the pin the esophagoscope unrolls
the spring The point slides beyond on the
esophageal mucosa without causing in-
jury (a) Once the pin has been straight-
ened out it can be withdrawn either
through the lumen of the esophagoscope
or as the tube itself is withdrawn (b)

which is evidence both as to principle and actual details of the complexity of the problem. The majority of these methods are exceptional procedures, a small number of which provide a solution for almost all the cases. Five methods are described here.

1 *The Procedure of Elongation* This is a sure method frequently used. It cannot be applied except when the length of the pointed end is less than the maximum diameter of the esophagus and when the end with the catch on it can be made to enter the extremity of the esophagoscope. During the course of this preliminary maneuver, before the actual extraction, the safety pin is held firmly

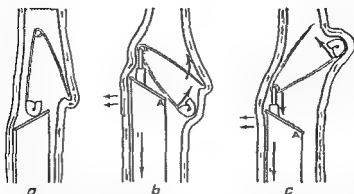
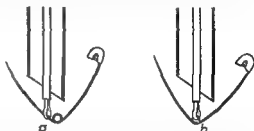


FIGURE 330 : Extraction of a safety pin (version in the plane of the widened ends) The esophageal wall is pushed aside by lateral pressure with the esophagoscope in order to permit grasping by means of the traction forceps applied to the ring formed by the spring (b) Traction effected by the forceps produces turning of the pin the closing end of which usually presses a point (A) of the ferrule on the end of the esophagoscope (b and c) (After Chevalier Jackson)

FIGURE 331 : Extraction of a safety pin a Incorrect grasp b correct grasp to avoid the escape of the pin



by the forceps without exerting any traction from above. The unbending of the spring is brought about by pushing the esophagoscope downward. In this manner the point of the pin slides along the mucosa in a retrograde fashion and does not cause a perforation (Fig. 329).

2. *Turning of the Pin in Its Plane of Expansion* This method succeeds in the majority of instances even when the pin is large and has a rigid spring. Its application is however rather treacherous and demands great attention to detail. The pin is grasped by a rotation forceps applied to the center of the ring which forms the spring and the version is brought about by pushing the esophageal wall aside with the forceps. This is often facilitated by counter-pressure with the esophagoscope upon the closing arm of the pin or by a simple point of support which serves as an axis upon which the pin may swing (Fig. 330).

3. *Endogastric Version* This method is indicated occasionally for the removal of a large pin with a stiff spring. Before attempting it one must make certain that the bite of the rotation forceps is applied in the ring which forms the spring and not on one of the arms (Fig. 331).

The pin is usually pushed into the stomach without any difficulty, where several jerking motions of the forceps cause it to turn around, whereupon it can be extracted with the point trailing behind without much risk (Fig. 332).

4. *The Method of Engaging the Point* This technique is very efficacious and often succeeds even with large pins with stiff springs. Only the point is engaged in the esophagoscope, the branch with the clasp on it, whose extremity

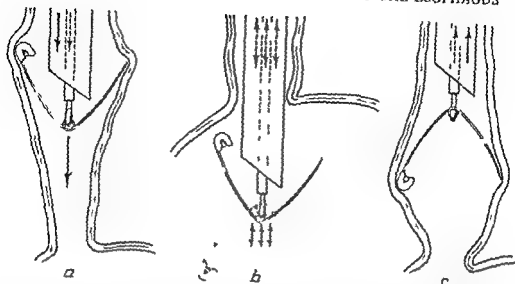


FIGURE 332 Extraction of a safety pin (endogastric version). The pin held by its spring (a) is pushed into the stomach. Several sharp to and fro movements of the forceps assures turning of the pin within the stomach (b) which permits the extraction of the foreign body with its point trailing behind (c).

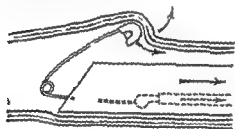


FIGURE 333 Extraction of a safety pin. Method of ensheathing the point. The clasp the surface of which is rounded and smooth slips over the esophageal mucosa, thus making it possible to remove the object without harm.

is rounded and smooth, remaining outside of the instrument during the extraction (Fig. 333).

5 *The Method of Closing the Pin Before Extraction* This technique is accomplished by coordinated movements of the forceps and the end of the esophagoscope or by the use of special instruments such as the closer with a movable ring or closing forceps several models of which are available. These instruments are relatively large, sometimes difficult to use and of more theoretical than practical value.

Extraction of Metallic Foreign Bodies by Jointed Magnets

Every metallic foreign body made of iron or steel can be removed with a magnet which has a flexible extremity (Fig. 334). This flexibility of the magnet makes its introduction along the walls of the esophagus possible without causing injury. The appliance is made by the General Electric Company. It is necessary first to determine whether the object to be extracted is made of iron or steel and in this regard all assertions of the patient and his relatives as well as the submission of a presumably similar object for comparison may be inaccurate. Investigations have been undertaken by Equen who has employed an instrument manufactured by the Waugh Laboratories for the detection of mines. When this instrument is placed on the surface of the skin as close as possible to the ingested foreign body, a buzzing noise is heard if the object is made of iron.

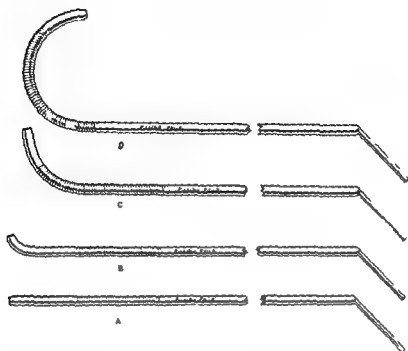


FIGURE 334 - Various shapes of magnets with flexible stems for the extraction of foreign bodies made of iron *A* Straight magnet *B* magnet with short curve *C* one with the mobile portion forming a right angle *D* another with an angle of 180 degrees (Chevalier Jackson)

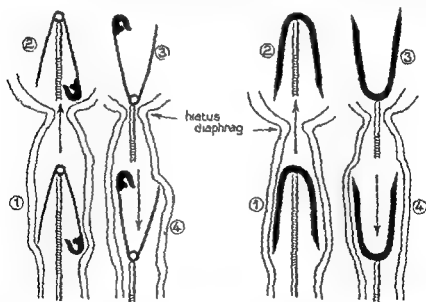


FIGURE 335

FIGURE 336

FIGURES 335 and 336 - Extraction of foreign body made of iron by a magnet with a flexible stem Endogastric version of a safety pin lodged point uppermost using an articulated magnet (Figure 335) Endogastric version of a staple by the same technique (Figure 336) (Chevalier Jackson.)

If no result is obtained, a heavy magnet such as that of Sweet or of Haab used in ophthalmology may be employed

With the patient lying on a fluoroscopic table, the core of the electromagnet is placed on the surface of the skin as close as possible to the foreign body. The shadow of the object is examined under the screen in the visual axis of the observer 90 degrees from an imaginary line which goes from the axis of the magnet to the foreign body. If the latter is made of iron or steel, it is attracted by the magnet when the current is turned on and returns to its former position with the interruption of the current.

TECHNIQUE OF EXTRACTION The patient is placed on the fluoroscopic table in the Chevalier Jackson position. The flexible magnet is introduced into the right pyriform sinus which is usually better seen than the left. The laryngoscope is removed and with great caution contact of the instrument with the foreign body is established. A physician skilled in endoscopy will not need a laryngoscope to visualize the pyriform sinus. The foreign body is then slowly withdrawn and if it becomes detached during passage through the mouth, contact is re-established.

In the case of a safety pin lodged with the spring uppermost the withdrawal should be watched closely to avoid perforation of the esophageal wall. If on the contrary the point faces upward, the pin should not be removed when contact is first established. It is better to employ the magnet to turn the pin in the stomach according to the technique of Chevalier Jackson.

Once the pin has been reached by the magnet, it is directed cautiously into the stomach and guided by appropriate manipulations along the gastric wall. As soon as the magnet makes contact with the end which carries the spring, the pin is withdrawn slowly in such a way as to permit continuous contact with the magnet. If a separation occurs during the passage through the esophageal hiatus of the diaphragm, the pin falls into the stomach and the maneuver must be repeated. If separation occurs in the esophagus, immediate contact must be made again. All manipulations must be carried out with gentleness (Figs 335 and 336).

Equen and Hollinger employ the forceps if the pin escapes from the magnet. There is reason to disagree with this practice because endogastric extraction presents several advantages. The walls of the stomach are thick and less liable to perforation than the esophagus; there is ample room in the stomach and the stomach may be insufflated with a rubber bulb to provide more room.

Staples may likewise be turned with a flexible magnet in the same manner as with the forceps in the Chevalier Jackson technique (Fig. 336).

If an iron object or a pin is found in the stomach of the patient upon his arrival at the clinic and if the use of a straight flexible magnet does not succeed, a curved magnet may be employed (Fig. 334, B, C, D). The curve of this instrument straightens out during its passage through the esophagus but reforms again as it emerges from it.

Extraction of Foreign Bodies from the Mouth of the Esophagus

In practice the foreign bodies which are most difficult to extract are those in the mouth of the esophagus and in the cervical segment. These are usually

flat objects like coins which become lodged in the transverse plane lying close to the posterior wall and are often hidden under the bulge made by the contracted cricopharyngeus muscle. In addition, edema and thickening of the mucosal folds, which are particularly well developed in certain subjects, often add to the difficulty. After locating the foreign body with a roentgen film the sphincter is passed with more or less difficulty, using one's favorite esophagoscope. Usually the instrument goes beyond the foreign body without its being seen. This may occur for the reasons just mentioned or because the pressure of the end of the esophagoscope as it is being introduced may push up a mucosal fold which completely obscures the object (Fig. 312, page 473).

A further difficulty arises from the use of an esophagoscope of standard length. With a foreign body engaged in the upper esophagus the major portion of this instrument remains outside the mouth, where it does no good. In addition, it is necessary to employ long instruments to suit the length of the tube. For these reasons it is preferable to employ a laryngoscopic speculum, the light source of which is at the distal end. The speculum is introduced, the larynx is identified as soon as the arytenoids are recognized, and the speculum is slid along the wall of the pharynx behind the arytenoid fold. It is then drawn forcibly forward. In this way the mouth of the esophagus is visualized. The cricopharyngeus muscle is pushed aside and the contracted muscular bundle is flattened, often with the aid of Adrenalin to shrink the edematous mucosa. The posterior wall of the cervical esophagus against which the foreign body, especially if it is a coin, lies is thereby exposed.

This procedure employed in numerous instances has always given satisfaction. It is the method of choice in the exploration of the mouth of the esophagus and by its means in one interesting case it was possible to extract easily a bony fragment made up of three vertebrae and a rib of a rabbit (Terracol).

Surgical Procedures for the Extraction of Complicated Foreign Bodies

Provided the patient is seen early by someone who is skilled in the management of such cases, a foreign body lodged in the esophagus can usually be extracted perorally. There are, however, complicated situations which may necessitate the application of surgical techniques for the extraction of the object. These are as follows:

1. A foreign body impacted in the esophagus for several days with resulting infection in the neck, a Minnigerode sign, induration of the neck, and exquisite pain along the carotid sheath. This situation is particularly common with small pointed bones which penetrate the mucosa or metallic objects with sharp points or edges. The various aspects of such cases were presented in the chapter on perforations (Chapter 22).

2. Large objects impacted in the thoracic esophagus for several days, usually at the lower end.

3. Foreign bodies which are unusually traumatic, like razor blades.

4. Foreign bodies which have failed to respond to the usual methods of extraction and still remain in the esophagus.

With present-day anesthesia techniques and the use of antibiotics, healing is the rule.



FIGURE 337 Lesion provoked by prolonged lodgement of a foreign body within the esophagus

CERVICAL ESOPHAGOTOMY This operation conceived by Verdué for the extraction of a foreign body lodged in the cervical segment was carried out on a living patient by a Limousin physician named Goursaud in 1778. Although ill-received at first and reserved only for cases in which the object was actually protruding through the wall of the organ, it was revived by Terrier as an emergency procedure. Its indications now, however, are limited because of the popularization and perfection of endoscopic techniques for the removal of foreign bodies through the natural passageways.

Indications The almost exclusive indication is the necessity for the removal of a complicated foreign body which cannot be extracted in any other way. It should be considered whenever the object cannot be withdrawn perorally or pushed down into the stomach. It is usually necessary only when the foreign body is unusually large or irregular in shape or has been lodged for three or more days without attempts to remove it, in which case the situation is complicated by the presence of emphysema and the evidences of infection. The signs of infection which should prompt the operator to abandon further attempts by the peroral route are edema of the walls of the laryngopharynx, diminution of laryngeal crepitation, deep induration, subcutaneous emphysema, retrotracheal pain, painful deglutition, and a fetid breath. In addition there may be fever, rapid pulse, or dyspnea. The patient may appear toxic. Any or all of these manifestations demand the immediate performance of an esophagotomy not only to remove the offending object but also to aerate and drain widely all the fascial spaces of the neck.

Figure 337 illustrates the extent of local injury within the esophagus which

may result from prolonged delay or failure to remove the object. Such complications can be prevented by prompt recourse to extraction by esophagotomy in every instance when the foreign body does not yield to endoscopic extraction.

Operative Technique The patient should be placed on his back with a cushion under the shoulders and his face turned to the side opposite the surgeon (usually to the right).

Local or regional anesthesia may be employed, but if the services of a skilled anesthetist are available endotracheal inhalation anesthesia is preferable.

The incision is made over the anterior border of the sternocleidomastoid muscle from the sternoclavicular joint to the upper edge of the thyroid cartilage. For anatomical reasons the left side is chosen except when the foreign body

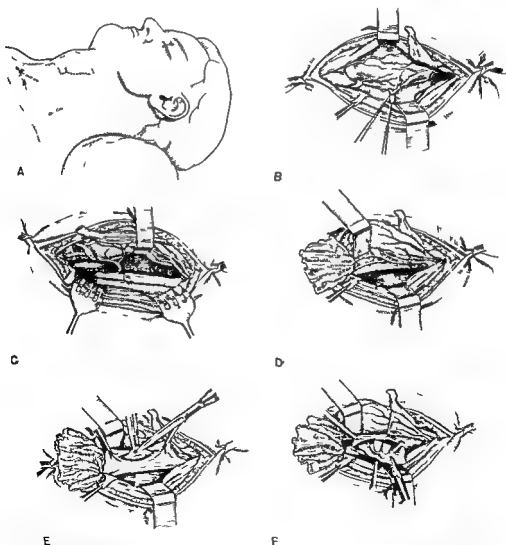


FIGURE 338 Drawings illustrating the stages of external esophagotomy for the extraction of a foreign body. *A* Line of incision. *B* Omohyoid muscle divided. Thyroid veins tied. *C* Retraction of the thyroid gland exposing the inferior thyroid artery for division. *D* Esophagus clearly exposed. Gauze packing to wall off the superior mediastinum. *E* Incision of the esophageal mucosa after incising the muscular coat. *F* Extraction of a large denture through the opening.

bulges through the right side or when there is a right-sided cervical abscess (Fig 338, A) The skin, platysma muscle, and superficial fascia are divided. The sternomastoid muscle is retracted posteriorly.

The deep fascia is incised and the omohyoid muscle is severed. The carotid sheath is retracted posteriorly (Fig 338, B). The left lobe of the thyroid gland and the sternohyoid and sternothyroid muscles are retracted anteriorly. The inferior thyroid artery and several thyroid veins may have to be divided and tied (Fig 338, C). The esophagus is then exposed and freed from the prevertebral fascia by a sweep of the finger or the blunt edge of the scissors (Fig 338, D). If the foreign body is bulging, the incision is made directly over it. If the object is obscured, the incision is made over the place where it is thought to be. A traction suture or a hemostatic forceps is applied to the lateral wall of the esophagus above and below the place where the foreign body is lodged. A longitudinal incision is made through the esophageal wall between these two points (Fig 338, E).

Once the esophagus is open, the incision may be enlarged lengthwise to the point where the object can be extracted without lacerating the tissues (Fig 338, F).

If because of edema and inflammatory thickening the esophagus cannot be identified, a large soft bougie may be inserted from above to the point where the foreign body lies and the incision made over the instrument as it is palpated from the exterior.

If the foreign body is lodged in the hypopharynx, the incision must be enlarged upward and the superior thyroid vessels divided and ligated. The inferior constrictor muscle of the pharynx is incised, the mucosa opened, and the object exposed.

If on the other hand the foreign body is located in the superior mediastinal segment, it can usually be dislodged with the aid of the finger or extracted through the esophagotomy incision using a suitable forceps.

In the absence of obvious infection the incision should be closed longitudinally with a layer of fine silk sutures in the mucous membrane and another similar layer to approximate the muscle edges. Under the protection of suitable antibiotics the incision may then be closed with a layer of sutures in the platysma muscle and another in the skin. If there is gross infection, however, or if the esophagus is lacerated and the edges of the rent ragged or inflamed, no effort should be made to close it. The wound is treated in that case as for a perforation of the esophagus in the cervical region (Chapter 23).

Aftercare It is undesirable to insert an indwelling nasogastric tube, especially in the presence of infection, because of the danger of causing ulceration.

Feeding, hydration, and care of the wound follow the rules set forth in Chapter 23 (perforations).

Ultimate healing can be expected and stricture formation is unusual even when a primary closure cannot be performed.

THORACIC ESOPHAGOTOMY In the rare instance when a foreign body cannot be extracted from the thoracic portion of the esophagus below the aortic arch or pushed on into the stomach, surgical intervention becomes necessary. A standard thoracotomy incision is employed. The esophagus is opened longi-

tudinally over the impacted object. Extraction of the foreign body and closure of the esophagus follow the principles outlined previously. Wound closure and aftercare are identical with those described under thoracotomy for treating perforations (Chapter 23).

TRANSABDOMINAL ESOPHAGOTOMY : With an object lodged in the cardiac end of the esophagus, the upper abdomen may be opened and the foreign body removed through a gastrotomy. Immediate closure of the stomach is made by the usual techniques.

It should be emphasized that the decision in favor of active surgical intervention in any of these three areas must be made without delay and before the development of infection, which vastly complicates the problem or may render its successful solution impossible.

CHAPTER 26

Benign Tumors

BENIGN tumors of the esophagus are rare. Statistics vary, but their relative infrequency is attested by the report of Calmenson and Clagett of only 44 in 7,459 autopsies. In the living Moersch and Harrington found 59 benign esophageal tumors as the cause of dysphagia among 18,459 patients who complained of that symptom.

The separation of benign tumors into two groups according to whether or not they are sessile or pedunculated is based more on their morphological and clinical aspects than on their pathological characteristics. Tumors of various histological types may be found in either group. With polypoid or pedunculated tumors the location and size, because of their mechanical influence, comprise the only important differences.

Pedunculated tumors arise at the superior or inferior orifice of the esophagus. Because of their great mobility they present characteristic signs.

Sessile tumors may be encountered at any level along the entire length of the esophagus. Even when they reach a considerable size, they may remain silent and are discovered only at autopsy. Sometimes they give evidence of their presence by causing obstructive symptoms. Their etiology is unknown. Cysts may occur in early infancy. Polyps are encountered principally in men over forty years of age.

Pathological Anatomy

Various types may be observed including cysts, leiomyomata, neurofibromata, fibromata, hemangiomata, papillomata, and mucosal polyps. The most frequent is the leiomyoma. Moersch and Harrington in a series of 43 benign tumors found

Leiomyomata	32
Hemangiomata	3
Papillomata	3
Cysts	2
Polyps	2
Neurofibromata	1

Tumors of Epithelial Origin

CYSTS Cysts of the esophageal wall are either acquired as a result of occlusion of the duct of an esophageal gland (retention cyst) or, more often, they are congenital and are then spoken of as reduplication cysts. They may be discovered accidentally when small (pea-sized or slightly larger), but they become relatively large (3 to 4 cm in diameter or larger) before they begin to cause symptoms. Although usually single in occurrence, they may be multiple. They arise in the submucosa where they form a small pouch filled with clear or slightly viscid fluid. Bronchogenic cysts contain the characteristic stringy mucus.

Histological examination reveals in the first type a lining epithelium of more or less flattened cells depending on the degree of distention of the cyst. The other layers of the cyst wall are formed of bundles of connective tissue which fuse insensibly with the tissues of the region in which the cyst develops.

Cysts of the congenital type seen usually in the lower esophagus or at the level of the tracheal bifurcation contain a characteristic milky, or gelatinous type of fluid. They may be classified as the bronchogenic or the esophageal reduplication (or enterogenous) type according to the histological characteristics of their lining membrane. The lining of the former is made up of stratified epidermoid epithelium with cilia. The latter are lined with epithelial cells similar to those found in the normal esophagus. Others have linings which give the appearance of gastric mucosa. Those of this type, when they develop in the upper esophagus, may arise possibly in islands of gastric mucous membrane or in the so-called superior cardiac glands of the esophagus. Both of these types are undoubtedly vestigial remains of a reduplication of the primordial foregut of the embryo.

PAPILLOMATA Papillomata resembling those found in the skin or in the oral or anal mucosa may be encountered. A frequent finding at autopsy in

FIGURE 339 Photomicrograph of a papillomatous polyp of the esophagus ($\times 25$). This polyp whose pedicle is at *d* is actually made up of three secondary polypoid formations (*a b c*). The epithelial layer is thinned out on the surface of the tip whereas at the base it is thickened and papillomatous where the layers of two adjacent polypoid components become fused. The vascular connective tissue stroma is infiltrated with lymphocytes due to chronic inflammatory changes.

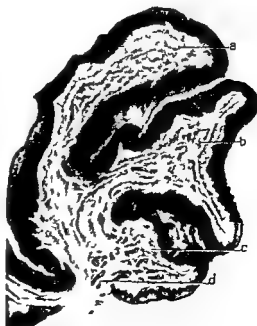




FIGURE 340 Photomicrograph of a papillomatous polyp ($\times 15$) At *a*, papillomatous proliferation of the Malpighian layer, *b*, dilated blood vessels of interpapillary crests engorged with red blood cells *c* richly vascularized chorion *d* epithelial appendage of the polyp consisting merely of large acanthotic process

elderly subjects, these tumors appear as small isolated or conglomerate protuberances the size of a lentil or the head of a pin. They are covered by a whitish, thickened epithelium. They resemble cutaneous warts (Fig. 339). They may be the result of chronic surface irritation of the esophageal mucosa.

Histologically these papillomata are hypoplastic excrescences protruding from the epidermoid epithelium, characterized by hyperplasia of the epithelium and the subjacent vascular connective tissue (Fig. 340). There is often a superimposed acanthosis which implies a hypertrophy of the Malpighian corpuscles of the mucous membrane with leucoplakia. It is known that this lesion arises in dermopapillary mucous membranes from hyperkeratosis, with the abnormal presence between the cornified layer and the Malpighian bodies of a granular layer due to the accumulation of keratohyaline and eleidine granules which diffuse throughout the cornified layer.

Papillomata of the esophagus may become malignant. Such tumors which are rare, have been referred to as "adeno-acanthomata."

ADENOMATOUS POLYPS Another unusual tumor of epithelial derivation is the adenomatous polyp. From the histological point of view these tumors resemble the adenomata sometimes seen in any glandular membrane. There is a hyperplastic process involving simultaneously both the epithelial and the mesenchymatous tissues which form the glandular acini (i.e., the covering epithelial cells, the subepithelial membranes, and the vascularized connective tissues beneath). The abnormal growth of these two tissues does not always proceed in a parallel manner. It follows that the histological aspects vary depending upon whether the hyperplastic process predominates in either one or the other. Thus one may encounter

An *acinous adenoma* in which is seen a multiplication of the gland tubules with thickening of the subepithelial membrane,

An *intracanalicular vegetative adenoma* in which, as if the hyperplasia of the hyaline membranes were in advance of that of the epithelium, one sees the heaping up of the glandular lumina by the underlying layer in which there is

overgrowth of the supporting vascular connective tissue, while the epithelial investment is pressed upon by these outcroppings of the mesenchyma and becomes atrophied, sometimes substantially so

A *papillary adenoma* characterized by papillary epithelial vegetations which more or less obstruct the lumina of the glandular tubules, and finally

A *pseudo-acinous adenoma* in which the endocanalicular epithelial elements constitute in themselves pseudoglandular lumina These are regarded by some authors as premalignant lesions

Much of the time these diverse forms of adenomatous hyperplasia are intimately intermingled in a given portion of a tumor even in a single microscopic field

As with papillomata, the benignity of an adenoma depends upon the parallel if not actually synchronous growth of the epithelial and mesenchymatous tissues When these conditions do not prevail one of the factors in the development of carcinoma is realized

Tumors of Nonepithelial Origin

FIBROMATA along with leiomyomata comprise the majority of benign tumors of the esophagus They develop in the submucosa and are therefore intramural In the beginning they elevate the mucosa forming small localized swellings at first no larger than a pea, but their volume and above all their length may increase to the extent that they become pedunculated Sometimes the stalk is so long that they reach or even pass beyond the cardia Their consistency is elastic The overlying mucosa remains normal but it may become ulcerated in places because of interference with blood supply or because of mechanical factors caused by their length These tumors are usually vascular and are analogous to the fibromata seen in the pharynx

From the histological viewpoint they may be simple or undifferentiated The former are tumors which result from the autonomous proliferation of connective tissue which becomes differentiated in its adult form They are made up of well formed, mature cells the cytoplasm of which is rich in collagen fibers These fibroblasts are grouped in whorl-like bundles surrounding a blood vessel (Fig 341) There are no obviously abnormal cells nor is there evidence of invasiveness Fibromata push aside but do not infiltrate the surrounding tissues

FIGURE 341 Photomicrograph of a fibroma of the esophagus ($\times 130$) represents connective tissue bundles cut in various places In the left lower corner is a sheet of fibrous tissue poor in fibroblasts with very dense bundles of collagen





FIGURE 342 Drawing showing the gross appearance of a leiomyoma of moderate size bulging into the lumen of the esophagus (After Haslinger)

The incompletely differentiated fibromata have the same structure but they are poor in collagen fibers and relatively rich in fibrocytes

MYOMATA Leiomyomata are by far the most common tumors arising from the muscle layers. They may occur singly or be multiple or conglomerate. They vary greatly in size from 1 cm. or less to 10 cm. or more in diameter (as large as or larger than a goose egg). They are seen usually in the lower third of the esophagus, but they may be found in the upper third as well. They often narrow the lumen markedly but usually spare the cardia. They may or may not be pedunculated.

When single they are simple benign tumors such as may be encountered in any organ (Fig. 342). They are found often only at autopsy, having caused no symptoms during the lifetime of the patient (Fig. 343). When multiple there is room for speculation regarding their cause. There may be, as Lortat-Jacob proposes, a predisposition leading to a diffuse nodular myomatosis. Some leiomyomata tend to develop in a circular fashion (Fig. 344). This may be explained by the fact that the majority of the tumors of this sort arise from the smooth muscle tissue of the circular muscle layer.

The smooth muscle fibers of which they are made up show both hypertrophy and hyperplasia without any modification of structure. As with fibromata a whorl-like arrangement of the fibers is often seen, but the cellular elements are provided with more or less abundant contractile fibrils.

Sometimes these fibrils may become atrophic or even disappear while the collagenous portion of the smooth muscle cells becomes hypertrophic with a resulting tendency to become fibrinous. This is a well known pathological process affecting smooth muscle whether in the uterus, esophagus, stomach or any other organ composed of this type of tissue. As with uterine myomata, leiomyomata of the esophagus also may undergo calcification (Fig. 345).

In diffuse myomatosis of the esophagus, histological studies have shown a considerable modification of the esophageal musculature involving the transverse or circular muscle layer with a more or less striking thickening due to the juxtaposition of a great number of rather limited small nodular elements

The possible etiological association of hypermotility and the development of leiomyomata has been the subject of considerable speculation. This provides a possible explanation for the frequent occurrence of these tumors in the sphincter like lower portion of the esophagus. The occasional occurrence of a diverticulum proximal to the tumor bespeaks also the possible presence of some

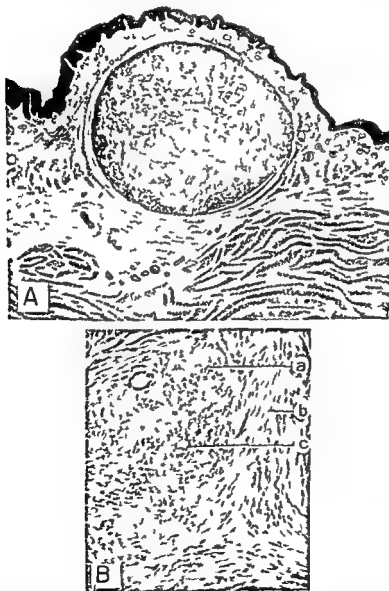


FIGURE 343 · Photomicrograph of *A* small leiomyoma of the esophagus showing its intramural development. *B* higher power section ($\times 110$) showing bundles of smooth muscle fibers sectioned transversely at *a*, longitudinally at *b*, *c* is a blood vessel (*A* After Fischer)



FIGURE 344 Diagram showing the manner in which a leiomyoma may grow circumferentially around the esophagus from a single point of origin in its wall (Made from the specimen removed in operation on the patient whose esophagogram is shown in Figure 345)



FIGURE 345 Esophagogram of a patient with a calcified esophageal leiomyoma growing circumferentially around the esophagus (Fig 344) Note the barium filled traction diverticulum produced by the outward enlargement of the tumor 1, Calcified leiomyoma 2, traction diverticulum

muscular dyskinesia, whether it be cause or effect. It is difficult to understand why some patients, even in the presence of large smooth muscle tumors, experience no symptoms whereas others have a considerable degree of dysphagia.

RHABDOMYOMATA are exceptionally rare tumors occurring in the upper third of the esophagus where some of the muscle is of the striated type. They are usually sarcomatous (see Chapter 27).

LIPOMATA are exceedingly rare in the esophagus. They may develop either in the submucosal or in the muscular layers. They are usually single but have a tendency to become pedunculated. Like lipomata elsewhere, their structure reproduces exactly that of normal adipose tissue consisting of vesicular fat cells, the multiplication of which gives rise to lobules separated by partitions of vascular connective tissue. They arise from the hypertrophy of lipoblasts which are small elements similar to fibroblasts but with a cytoplasm filled with droplets of fat which may be demonstrated by special techniques or which are dissolved

by the reagents used in the usual staining methods giving rise to the areolar appearance seen on histological sections (Fig 346)

MYXOMATA are rarely observed in the esophagus. They develop at the expense of the embryonic mucous tissue with more or less branching or star-shaped cells which are interlaced by their cytoplasmic extensions in the midst of a basic substance presenting the histological reaction of mucus (Fig 347)

HEMANGIOMATA Hemangiomas or hemangio-endotheliomas result from localized hypertrophy of the blood vessels of various sizes (Fig 348). In some instances there is an associated hypertrophy of the connective tissues which gives rise to hemangiofibromata. They may be pedunculated. They may also be a part of multiple hemangiomatosis of the alimentary tract (Rendu Osler disease) (Chapter 12)

LYMPHANGIOMATA have been reported in a few instances. Ferguson and Hackworth observed such a tumor proved by biopsy in a seven months old infant. The tumor disappeared after treatment with roentgen rays.

MESENCHYMATOUS TUMORS of the esophagus are rare. They arise intramu-

FIGURE 346 Photomicrograph of a section from a lipoma removed from the esophagus ($\times 120$). *a* Fat cells *b* connective tissue partition.

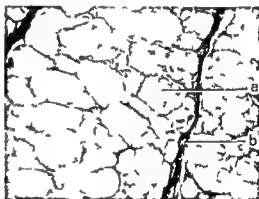


FIGURE 347 Photomicrograph of a section from a myxoma of the esophagus showing myxomatous tissue rich in branching cells and blood vessels ($\times 130$).



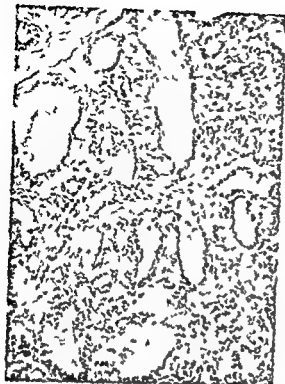


FIGURE 348 Photomicrograph of section from an hemangioma of the esophagus showing blood vessels in abnormal numbers cut in various planes ($\times 130$)

rally beneath the epithelium. They may be benign or malignant. The polymorphism of these tumors is interesting. They are made up of several tissues of mesenchymatous origin: reticulo-endothelial tissue, areolar connective tissue, adipose tissue, myxomatous tissue, cartilage, or even bone or muscle. Sometimes one type of tissue predominates, but it is never single. The other mesenchymatous tissues always participate in the neoplastic proliferation and contribute to the polymorphism which differentiates these tumors from those which comprise a single cell type. They are sometimes called 'mixed tumors' and bear a resemblance to those of similar nature which occur in the salivary glands.

Any of the above described benign tumors of the esophagus may be found in a pedunculated form in which they may be small or very large and may have a short or a long stalk. Depending upon their nature they may be soft or hard, smooth or lobulated and, depending upon their blood supply, they may have an intact or an ulcerated surface.

Clinical Characteristics

When the tumor is small and sessile it is invariably asymptomatic and may never be discovered except post mortem. If it is large the tumor, especially if it arises from the upper portion of the esophagus, may cause bouts of coughing interference with deglutition, nausea, regurgitation or simply progressive dysphagia especially for solid food, ending in complete obstruction when the tumor finally blocks the esophagus. Papillomata are usually fungating and provoke varied difficulties, principally a sensation of stoppage of food during swallowing.

When the tumor is pedunculated, particularly when attached just below the inferior margin of the cricoid cartilage in the midline of the anterior wall

of the esophagus, it causes characteristic troubles depending upon its size. If small there may be no symptoms. If large, cervical or thoracic pain may be felt and nausea, regurgitation, and progressive dysphagia develop. Sometimes during vomiting, such a tumor swings up into the mouth where it is felt by the patient but on swallowing it disappears into the esophagus. If the stalk is 15 cm or more in length the tumor may actually become extruded from the patient's mouth during fits of coughing or attacks of vomiting. If somewhat shorter, the stalk may allow it to reach no further than the pharynx where it may lodge in the upper part of the larynx and cause asphyxiation.

These tumors may be seen at will by stimulating the vomiting reflex.

Roentgen and Esophagoscopic Examinations

ROENTGEN EXAMINATION discloses an obstructing or partially obstructing obstacle. With the sessile intramural type of tumor the barium passes over and around the mass and because of the intraluminal bulging of the tumor gives rise to a characteristic shadow with a dense periphery and a thinned-out center (Fig. 349, A).

ESOPHAGOSCOPIC EXAMINATION may lend confirmatory diagnostic evidence. It is useful to disclose polypi (Plate III, 8, and Fig. 350) but with smooth muscle tumors and intramural fibromata it is of relatively little benefit.



FIGURE 349 A Roentgen appearance characteristic of a benign intramural tumor of the esophagus showing cascading of the barium around the margins of the mass as it bulges into the lumen. B Photograph of the tumor (leiomyoma) removed by esophagomyotomy.

CHAPTER 27

Malignant Tumors

UNDER the heading, "Malignant Tumors," should be included not only carcinoma but also the sarcomata and certain other rare malignant tumors such as adeno acanthoma and melanotic sarcoma (melanocarcinoma)

Etiology

Tumors of Epithelial Origin

The frequency of occurrence of carcinoma of the esophagus relative to all other forms of malignant neoplasms is still unknown. Reported figures vary from 4 to 18 per cent. In relation to other diseases of the esophagus, carcinoma is by far the most common. Guisez reported 1256 carcinomata in 2500 patients with esophageal complaints.

PREDISPOSING FACTORS Although the cause of the disease is unknown, certain facts regarding its incidence are of interest.

Sex Taken as a whole, carcinoma of the esophagus occurs predominantly in men. A compilation of statistics from various European and American sources indicates an incidence of approximately 88 per cent in men and 12 per cent in women. In the retrocricoid segment, however, the greatest frequency is among women.

Age Carcinoma of the esophagus is unusual under the age of thirty. Beyond this age its incidence increases with each succeeding decade, showing that it is predominantly a disease of old age. Sarcomata, on the other hand, may occur in childhood and among adults do not show this striking predominance among the aged.

The disease is observed among people of all social classes, with an apparently greater frequency among those who are given to alcoholism, excessive use of tobacco, and faulty dietary habits.

Race There are no reliable statistics regarding the racial incidence. The apparent infrequency of occurrence among the Arabs, for example, as compared

with the French may be the result of failure on the part of patients to consult the physician or of delay in making the diagnosis early after medical assistance has been sought

Heredit Nothing definite is known regarding a hereditary predisposition other than the common knowledge that development of malignant tumors of various organs and tissues tends to run in families. There is no evidence of a specific tendency to develop carcinoma of the esophagus alone

Antecedent Diseases Much has been written about the possible predisposing effect of syphilis or tuberculosis on the development of carcinoma of the esophagus but this is a useless speculation

Alcoholism The chronic irritation due to the local effect, aided possibly by the diminished resistance induced by malnutrition in chronic alcoholics, may mean that excessive use of strong alcoholic liquors has a predisposing influence which favors the development of carcinoma of the esophagus. Whether this is true or not, the disease is relatively common among those who use alcoholic beverages to excess

The positive effect of tobacco as a predisposing agent, though long suspected, has only recently been shown to have a valid statistical basis in studies made by the American Cancer Society

Trauma In line with the possible local injurious effect upon the epithelium of the mucous membranes is the effect of the habitual ingestion of large volumes of unusually hot liquids or food. There is, for example, a high incidence of carcinoma of the esophagus among the Chinese who eat their rice boiling hot

Inflammation and Stricture As with the development of a carcinoma of the skin in an osteomyelitis sinus, there is suggestive evidence that carcinoma of the esophagus may occur with greater frequency in areas of chronic esophagitis or cicatricial stenosis than in the normal mucous membranes. Isolated cases may be recalled in the experience of every observer (see page 151)

Chronic Obstruction It is possible that chronic stasis of food in the esophagus proximal to a partial obstruction exerts a carcinogenic effect over a prolonged period of time. This is suggested by the clinical observation of the relative frequency of occurrence of carcinoma in the esophagus of patients with mega-esophagus in whom the incidence is approximately 4 per cent (see Chapter 9)

Benign Tumors In rare instances primary benign tumors of the esophagus may evolve into carcinoma. These are the papillomata and adenomatous polyps. Occasionally a benign polyp is found in the presence of a carcinoma but it cannot be established that the carcinoma represents the malignant degeneration of a similar polyp

Carcinoma arising from other sources may reach the esophagus by way of blood borne metastases or by direct extension. The former are exceedingly rare. The latter are of frequent occurrence either at the upper end, into which a carcinoma of the pharynx may spread by continuity, or at the lower end where many cases of involvement by adenocarcinomata of the gastric cardia are seen



FIGURE 351 Gross appearance of melanotic sarcoma of the esophagus (primary) (Jaleski, Thomas and Valdo)

Tumors of Nonepithelial Origin

Primary sarcomata of the esophagus are rare tumors. They may occur at any age and are more frequent in men than in women. Histologically they are usually of smooth muscle origin (leiomyosarcoma), although in the upper third an occasional sarcoma arising from striated muscle tissue may be encountered (rhabdomyosarcoma).

Primary melanosarcomata of the esophagus have been reported in a few instances (Figs. 351 and 352).

Mixed forms are sometimes seen. These are usually called carcinosarcomata or adeno-acanthomata.

Pathological Anatomy

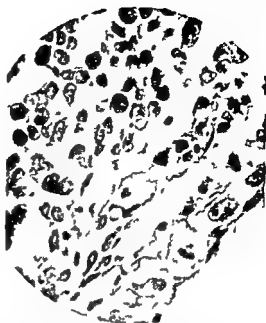
Epidermoid Carcinoma

The typical carcinoma of the esophagus is epidermoid in type.

LOCATION These tumors occur most frequently at or near the normally narrow segments of the esophagus. In a total of 1646 cases collected by Samaja the localization was as follows:

Upper third	266
Middle third	536
Lower third	700
Upper two-thirds	21
Lower two-thirds	48
Entire esophagus	6
Undetermined	49
TOTAL	1646

FIGURE 352 Photomicrograph of primary melanotic sarcoma of the esophagus showing histologic structure



If carcinomata arising in the cardia and invading the esophagus are excluded, the preponderance is in the middle third. In one author's experience the order of frequency of occurrence of epidermoid carcinomata in the various anatomical regions that are of technical importance to the surgeon is as follows: *middle portion, lower end, cervical segment, and superior mediastinal segment (R H S)*. If adenocarcinomata, many of which are gastric in origin, are included, the most common location is the lower end.

SIZE Great variations in size occur from small, often accidentally discovered tumors to enormous masses sometimes involving all or the major portion of the organ. The average size measured along the length of the esophagus at the time of discovery is 3 to 6 cm.

GROSS APPEARANCE AND SHAPE There are three general types according to their gross physical characteristics. These are (1) the fungating vegetative, polypoid type; (2) the ulcerating type; and (3) the infiltrating submucosal or scirrhous type. Of these the ulcerating form is the most common.

MULTIPLICITY OF TUMORS Although carcinoma of the esophagus usually occurs as a single tumor, there may be more than one or the growth may be associated with one or more tumors of another histological type or with a carcinoma of another organ such as the stomach.

The occurrence of two independent carcinomata of the same esophagus is not exceptionally rare. Sometimes these are widely separated, often one in the upper portion near the mouth of the esophagus and another at the lower end. The coexistence of more than two carcinomata is unusual. It is not certain whether multiple carcinomata result from intramural spread by way of the lymphatics or the implantation of exfoliated tumor cells from a single growth to other vulnerable sites. It is more likely that in the majority of instances they represent separate primary foci of the disease.

The coexistence of a carcinoma and a sarcoma at different levels in the

esophagus has been reported on several occasions, but this association is so unusual that it is probably merely a matter of chance occurrence

The finding of a leiomyoma in the segment of esophagus removed because of a carcinoma is not an exceptional occurrence (approximately 0.5 per cent of cases of esophagectomy for carcinoma [R H S]). As mentioned before (Chapter 26), benign tumors of epithelial origin such as polyps and papillomata may be found in the same esophagus with a carcinoma. Any histological relation between them, however, is not certain.

Coeistence of primary carcinoma of the esophagus and carcinoma of other organs is sometimes encountered. In order to exclude the possibility of relationship between them the tumors must be histologically characteristic of each of the organs involved. The most frequent association of this sort is an epidermoid carcinoma of the esophagus and an adenocarcinoma of the stomach.

Coexisting multiple carcinomata involving the esophagus and the colon, the kidney, and the lung are on record.

Carcinoma of the pharynx, palate, nasopharynx, epiglottis, tonsil, floor of the mouth, or tongue may be found in the same patient with a carcinoma of the esophagus.

COMPLICATING DISORDERS *Dilatation of the esophagus above a carcinoma* rarely assumes any degree of prominence. When it is pronounced, the probability is that the neoplasm has arisen in a mega esophagus or that the growth is of the slowly developing scirrhus type. In the latter event the dilatation is, however, never as large as in achalasia.

Perforation Perforations of carcinoma of the esophagus usually occur in the ulcerating type and are due in part to the invasive properties of the tumor and in part to the accompanying infection. Perforation occurring in the non-ulcerative type is usually the result of instrumentation with a bougie or the esophagoscope. Overzealous use of the biopsy forceps is a possible cause.

Spontaneous perforations leading to the formation of a fistula with the respiratory passages is a frequent occurrence. Involvement of the trachea is the predominant pattern. A collected series of cases derived from several sources shows the following relative frequency:

Fistula with trachea	144
Fistula with bronchi	95
Fistula with lung	60
Penetration of pleura	7

As mentioned in Chapter 21, when a bronchus is involved, the left side is more often affected than the right. The right bronchus is invaded usually only by a carcinoma arising in the dilated esophagus of achalasia.

A periesophageal abscess resulting from erosion of a carcinoma of the esophagus is usually found in relation to the thoracic segment. Such an abscess in the mediastinum may penetrate the lung to form an abscess of that organ as well, often leaving a fistula tract into the esophagus. There may be multiple intercommunications.

Erosion of the aorta is a fairly frequent complication of advanced carcinoma of the esophagus. The inevitable result is sudden death from overwhelming

hemorrhage Invasion of other arteries notably the carotid, innominate, subclavian, inferior or superior thyroid has also been reported Involvement of a pulmonary artery is almost unknown Penetration of large veins such as the superior vena cava or the pulmonary vein is even more unusual

SPREAD The spread of a carcinoma may be by direct propagation as when a lesion of the lower esophageal segment invades the cardia, by distribution through the regional lymphatic channels or by hematogenous dissemination

Lymphatic spread in the wall of the esophagus is so frequent an occurrence that carcinoma cells are often found in the intramural channels several centimeters beyond the obvious limits of the tumor *Metastases to regional lymph nodes* is a frequent finding in patients operated upon amounting to 75 per cent or more of all cases In the cervical segment the deep and superficial nodes of both sides of the neck as well as the paratracheal nodes in the superior mediastinum are frequently involved Carcinoma in the thoracic portion of the esophagus tends to spread to the paratracheal perihilar, subcarinal and lower periesophageal groups of nodes As the growth lies lower and lower in the esophagus there is an increasing tendency to metastasize to the subdiaphragmatic paracardial and left gastric groups of nodes With carcinoma of the middle and lower thirds, 70 per cent of those which give evidence of metastases show involvement of the subdiaphragmatic nodes This aspect of the behavior of carcinoma of the esophagus puts this form of neoplasm in the category of highly malignant tumors Figure 353 shows the lymph node groups to which esophageal carcinoma may spread

The scirrhus form of the disease appears to be the least likely to give rise to metastases in an early phase

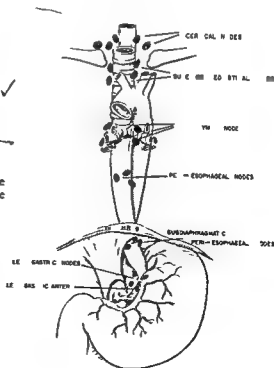


FIGURE 353 Diagram showing the lymph node groups which drain the esophagus

In some instances the metastases may give rise to symptoms before the presence of the primary tumor is even suspected

The *hematogenous spread* of carcinoma of the esophagus is ordinarily a late occurrence in the evolution of the disease. It may give rise to involvement of any organ or tissue of the body in order of frequency the liver, the lungs, the bones, the kidneys, the suprarenal glands, the brain, the heart, and the skin and subcutaneous tissues

Nonepithelial Tumors

About one-third of the sarcomata of the esophagus are polypoid. They may be very large (17 cm in length—Wegner). They may be multiple with two or three tumors at a time. The pedicle is usually small. Others are sessile and locally invasive. Metastases have been found in about one-half of the patients examined at autopsy.

In a series of 35 patients with primary sarcoma of the esophagus observed by Curtis, Smith, and Rusk, the average age was 53 years. There were 25 men and 7 women. The sex of the other three is not stated. Twenty-four tumors were located in the lower half of the esophagus, eight were in the upper third, in the remainder the location was not stated. As to shape, 14 were polypoid, 12 were nodular and diffuse, 9 were annular. Seventeen of the 35 patients died before the appearance of metastases. The trachea, the bronchi, or the lung was involved by direct invasion in seven.

The frequency of primary sarcoma of the esophagus in relation to carcinoma of that organ is shown in a series of malignant esophageal tumors reported by Resano and Albanese in which there was one sarcoma in 704 patients observed.

Histopathology of Malignant Tumors of the Esophagus

From the histological point of view, these tumors fall into two groups: the larger of epithelial origin (carcinomata) and the much smaller of mesenchymal origin (sarcomata).

Malignant Tumors of Epithelial Origin (Carcinoma)

Several histological types can be recognized. The first is the **TYPICAL EPITHELIOMA** spoken of as epidermoid or spindle-cellular carcinoma, the constituent Malpighian elements of which tend toward a process of keratinization forming keratotic epidermoid bodies. The cells are rich in connecting filaments (Figs 354 and 355). Others may be grouped as an **ATYPICAL EPITHELIOMATOUS TYPE** which derive from the basal cells and which do not undergo keratinization (Fig 356). A third form is **TRANSITIONAL**, in which both of the preceding types of cellular behavior are found in more or less close combination.

The usual carcinoma is a basocellular Malpighian epithelioma. This has led to the belief, formerly widely held, that carcinomata of the esophagus in general are not particularly malignant. It should be realized, however, that the difference between the malignancy of a typical epithelioma and the atypical

form is not as clear-cut as is ordinarily thought. In fact, the striking degree of abnormal mitotic activity of certain atypical epitheliomata imposes as much reserve regarding prognosis as this same karyokinetic activity observed for a typical epithelioma. These facts are reflected in the radiosensitivity of these tumors.

Among the less frequent histological types is the CYLINDRICAL EPITHELIOMA, which is very rare (19 out of 267 cases). The origin of these tumors is not exactly clear. They may arise in the esophageal glands themselves in ectopic islands of gastric mucosa, or in aberrant glandular inclusions in the esophageal mucosa.

However this may be the histological appearance varies according to the case from typical epitheliomata if they reproduce faithfully the pseudoglandular architecture, to papillary forms if the gland formation presents papillary proliferations in their lumina (Fig. 357) to pseudo-acinous if the intracanalicular epithelial hyperplasia produces pseudo-acinous cavities (Fig. 358) to atypical if the neoplasia develops in the shape of massive cords of cells, and finally to the mucoid type if the pseudoglandular groupings of cells show hypersecretion of mucus. These last are the colloid carcinomata of the older authors.

As always a minute study of the groupings of neoplastic cells in the deep planes of the section reveals that there is often a structure bordering on the



FIGURE 354 Specimen showing carcinoma of the esophagus. a Malignant ulceration with overhanging edges at b the organ f the proximal dilated portion of the esophagus above the tumor g diaphragmatic segment of the esophagus. The insert shows the histological structure of this carcinoma spindle cell Malpighian epithelioma with epidermoid whorls (h) ($\times 100$).

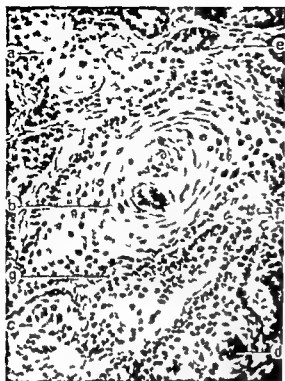


FIGURE 355 Photomicrograph of Malpighian or spindle cell type of epithelioma of the esophagus ($\times 120$)
a b, c d Tongues of malignant epithelial cells showing epidermoid whorls *e, f g*, new formed tumor stroma in the midst of the old stroma which has been pushed aside



FIGURE 356 Photomicrograph of a typical epithelioma spoken of as a basal cell type At *a b* and *c* tongues of malignant cells *d* focus of necrobiosis *e f* and *g* old vascular connective tissue stroma infiltrated by neoplastic lymphocytic plasmocytic reaction tissues (neostroma) ($\times 100$)

normal especially in the typical epitheliomata and their malignant nature is revealed only by the mitotic figures which they present

Furthermore, one must not forget that the different forms of cylindrical epitheliomata which have just been enumerated above may be seen in a single

neoplasm These carcinomata in fact are pre eminently polymorphic Whatever may be the variety of the epithelioma, whether of the stratified pavement type or the cylindrical there is one characteristic of their histological structure that deserves special emphasis, namely the *stroma* So far as the malignant tissue neoplasia is concerned, this stroma may be *preformed* (the paleostroma) with the role essentially of nourishing the tumor, or it may be *secondary* to the neoplastic process (the neostroma) and made up of lymphohistiocytic cells, the role of which appears to be that of resisting the grouping of cancer cells by the crea

FIGURE 357 Photomicrograph of section from a cylindropapillary epithelioma At *a* and *b* pseudoglandular spaces the lumina of which are obstructed by vegetations of epithelium and connective tissue *c* vascular connective tissue stroma containing inflammatory elements ($\times 105$)



FIGURE 358 Photomicrograph of section from a pseudo-acinous cylindric type epithelioma (adeno-acanthoma) of the esophagus at *a* *b* and *c* pseudo-acinous cavities in the very center of glandular pseudocavities *d* intercanalicular stroma ($\times 110$)



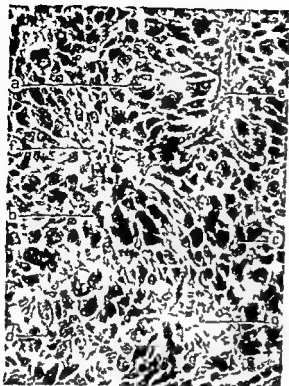


FIGURE 359 Photomicrograph of a section of an afibrillogenic fibrosarcoma. At *a*, *b*, *c* and *d* sarcoma cells of more or less large dimensions on the edge of blood vascular channels *e*, *f* and *g* ($\times 400$)

tion of physicochemical modifications of the medium which favor their abnormal growth

Therapeutically speaking, too much importance must not be attached to histological conclusions based upon the study of the stroma reaction. In fact, on the basis of a study of the histological structure of carcinoma of the esophagus, it is not possible to formulate an accurate estimate of the prognosis in terms of the clinical behavior of the growth or of its response to therapy

Malignant Tumors of Mesenchymal Origin (Sarcoma)

The usual type is a FIBROBLASTIC SARCOMA, formerly subdivided according to the shape of the cells into either the fusiform or the round cell varieties. These tumors arise from the mesenchymal elements or histioblasts which have a varied potentiality for development. They are frequently polymorphic, their mitotic and amitotic activity is considerable, their tendency to stimulate blood vessel formation is striking. The latter characteristic explains the presence of blood vessels that are deprived of ordinary walls and continuity (lacunae), which in turn explains the tendency of sarcoma cells to propagate by way of the blood stream (Fig. 359).

Sometimes a degree of fibril formation can be recognized in these sarcomata on the basis of which they may be subdivided into those which are fibroblastic or fibril-forming and those which are nonfibril-forming.

This concept of the presence or absence in the midst of the sarcomatous cells of a collagenous reticulum differentiated in the exoplasm of these cells is of great practical interest both from the point of view of diagnosis and from that of prognosis. In fact, according to Grynfeldt, who bases his opinion on precise clinical and anatomical correlations, the existence of a collagenous network sur-

rounding in its meshes the active parts of the neoplasm increases notably the cohesion of the sarcomatous tissue. This tissue *tends to diffuse* throughout the numerous lacunae which surround it and which communicate with the blood stream. Increased cohesion of this network apparently decreases the chances of blood-borne metastases. It follows that the fibrillogenic sarcomata appear to develop more slowly than the nonfibrillogenic type and that they are less likely to develop metastases.

RHABDOMYOSARCOMATA Sarcomata arising from the striated muscle fibers of the proximal one-third of the esophagus are encountered occasionally (approximately one in 1000 of malignant tumors). Histological identification of their nature depends upon the finding of the characteristic cell structure which resembles the striated muscle tissue from which they arise although such cells may be sparsely dispersed throughout a tumor tissue which is often pleomorphic in structure.

MYXOSARCOMATA Myxosarcomata of the esophagus are exceptionally rare tumors in a pure state. More often a myxoid structure represents one of the histological aspects of a fibroblastic sarcoma. This type of neoplastic tissue is characterized by a disorderly proliferation of star shaped or branching cells the prolongations of which intercommunicate. These cells are bathed in an edematous intercellular ground substance. Rarely they may be more or less rich in mucus which may be demonstrated by certain staining techniques. This type is actually a pure *myxosarcoma* in the true histological sense of the word.

OSTEOGENIC SARCOMATA have never been reported in the esophagus, but islands of cartilaginous or osteoid tissue may appear in a fibrosarcoma as a



FIGURE 360 Roentgen film of a patient with lymphoma primary in the esophagus. A Before roentgen therapy. B After three weeks of treatment showing complete disappearance of the tumor.

manifestation of the multiple potentialities of the mesenchymal cells from which such tumors are derived

LYMPHOBLASTIC SARCOMATA Lymphoma of the esophagus is a rare tumor (one case in six or seven hundred patients with cancer of the esophagus) [R H S] (Fig. 360). It develops from lymphoid cells which exist as rudimentary rests in the submucosal layer of the esophageal wall. A disorganized proliferation of lymphocytic and lymphoblastic elements with evidence of marked mitotic activity of the cells is characteristic of the histological appearance of these tumors.

Malignant Tumors of Mixed Histological Characteristics

These are malignant tumors which contain neoplastic tissues of several types. They are rare. Various terms may be applied, according to the preference of the individual pathologist, who may call them *epithelial sarcoma*, *adinosarcoma*, *mixed tumor*, or *adeno-acanthoma*. Whatever may be the histological structure presented by these tumors, one aspect is characteristic of them all. This is the multiplicity of anomalies of structure which they so often show, consisting of evidences of degeneration which are observed only in malignant neoplasms of the same histological type in other viscera.

Clinical Characteristics of Carcinoma of the Esophagus

The symptoms may be grouped in three periods: those of the onset, those of the full development, and those of the termination of the disease. This division, however, is didactic and arbitrary. The so-called period of onset is too often actually represented by the full-blown state of the disease. The first symptom in fact is usually a late manifestation.

Period of Onset

When the growth is small, consisting of a circumscribed island of neoplastic tissue, after a period of variable duration when the patient is totally unaware of its presence, the first symptom is usually a slight disturbance of deglutition. The patient begins to experience a transitory sensation of hesitation or arrest of solid food as it passes through the esophagus. This is almost invariably first noticed when swallowing a large particle of meat or bread. A mouthful of liquid swallowed soon after the ingestion of these foods usually overcomes the difficulty and the episode may be forgotten until the deglutition of the same type of material once again calls attention to the trouble.

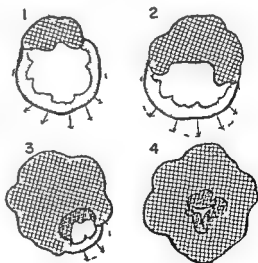
An observation which must impress everyone who sees many patients with carcinoma of the esophagus is that noticeable dysphagia rarely occurs until the disease has reached an advanced stage of development, with nearly complete circumferential involvement of the esophageal wall. This fact is explained by the peculiar ability of the normal wall of the esophagus to dilate or expand. As shown in Figure 361, it is only when three-fourths or more of the circumference is immobilized by the tumor that there is not sufficient distensibility of the remainder to permit the passage of even relatively large particles of food.

Later the dysphagia occurs with other foods until finally, as the obstruction progresses, even the passage of thick liquids (soup, etc.) becomes troublesome and the patient is reduced to the ingestion of clear liquids only.

ROENTGEN EXAMINATION in the early phase may show only a transitory arrest of the opaque mixture at the point where the growth lies, or the barium may pass through this area more slowly than usual, possibly because of the presence of esophagospasm. There may be no modification of the esophageal lumen. With the patient in the recumbent posture, the passage through the area of involvement is delayed because of the absence of the effect of gravity. Peristalsis in the zone of the lesion is then seen to be deficient, but one cannot make an exact diagnosis on the basis of the appearance of a segmental area of rigidity. If only a small amount of barium is given, minimal irregularities of the esophageal wall may be detected which are otherwise obscured. When the esophagus is full of barium at this stage, however, it is possible merely to make a presumptive diagnosis.

ESOPHAGOSCOPY. All patients with slight dysphagia, the exact cause of which cannot be discerned by roentgen examination, should have the benefit of an esophagoscopy in the hope of establishing a positive diagnosis by biopsy. In the early stage of the disease, the examination shows either a limited vascularized swelling or excrescence which bleeds easily, or a grayish ulceration (Plate III 5). The differential diagnosis between this and inflammatory spastic lesions is not always easy, especially in the lower third of the organ.

FIGURE 361 Diagram illustrating the reason why obstruction occurs so late in the course of the development of a carcinoma of the esophagus. 1 Early growth; arrows point to dotted line which indicates the degree of distensibility of the still uninvolved esophagus; few or no symptoms; transitory dysphagia on deglutition of very large bolus of food. 2 Growth involves one half or more of the circumference, leaving enough normal esophageal wall to dilate for the accommodation of all but the largest particles; patient still able to eat a reasonably normal diet, avoiding beefsteak, etc. 3 Almost complete encirclement by the tumor; patient reduced to the consumption of liquids but still able to swallow very soft solids with moderate difficulty due to the distensibility of the narrow strip of uninvolved esophagus. 4 Complete annular infiltration; no normal esophageal wall remains; patient unable to swallow any but the thinnest liquids; salivation; filling of the pyriform sinuses, etc. Note: In Stages 3 and 4, temporary improvement in swallowing ability is often experienced because of transitory enlargement of the lumen as a result of necrosis and sloughing of the center of the tumor (black cross hatching).



The Period of Development

The stage of full-blown development of the disease from the clinical standpoint appears more or less rapidly after the onset of the first symptoms which, as mentioned above, does not usually correspond with the onset of the disease itself. *Dysphagia* is the predominating symptom. It is progressive and permanent beginning with solids, then semisolids, then thick liquids. Finally, sometimes within one or two weeks, but in the average instance in approximately two months, the end stage of dysphagia is reached when clear liquids only may be swallowed. The patient is often aware of the location of the obstruction and will point to the region on the surface of the chest behind which the sensation of stoppage is felt. Sometimes he notices that he can ingest only a certain volume of fluid at one time before it is held up by the obstruction. An appreciation of the amount helps to estimate the relative location of the growth in the esophagus. As time goes on and dilatation of the esophagus above the tumor occurs, the amount of liquid which may be swallowed increases, but large degrees of proximal dilatation do not occur in the average case of carcinoma of the esophagus because of the relatively short duration of the obstruction.

A sometimes annoying symptom is *sialorrhea* which may be due in part to exaggerated secretion of the salivary glands, especially the parotid, as a reflex mechanism. This symptom is also a manifestation of chronic esophageal obstruction without specific reference to the cause. Severe degrees of obstruction augment the difficulty by causing the saliva to accumulate above the tumor whence it is regurgitated from time to time.

Regurgitation from the esophagus above the tumor of food previously swallowed occurs also on a reflex basis in an effort to avoid overdistention of the organ. Sometimes because of a feeling of discomfort evoked by the distention of the esophagus after eating, the patient himself will induce regurgitation by introducing his finger into the pharynx. With the exception of carcinoma developing in a mega-esophagus, the amount regurgitated at one time is never large. The material ejected sometimes contains bits of tumor tissue broken off from the growth.

A *foul breath* due to fermentation of food retained above the tumor or to necrosis of the growth itself usually is noticed late in the course of the disease, though it may be detected early when the tumor arises in an already dilated mega-esophagus.

Emaciation to the point of cachexia and developing rapidly may occasionally be the first sign of the presence of a carcinoma of the esophagus. Patients may lose as much as 5 to 10 kg (11 to 22 pounds) in several weeks. They live on their own tissue reserves from failure to obtain sufficient nourishment. Finally, when the deglutition of liquids becomes impaired, they begin to suffer dehydration which with the frequently associated sialorrhea renders them truly miserable.

Though not an early symptom, *pain* begins to be felt by the patient as the growth becomes large and produces a severe degree of obstruction. It is of two types. The first is intermittent, occurring during or just after deglutition, and is due to esophagospasm. It is felt in the substernal area at a level slightly higher than that of the tumor. As with any pain due to esophagospasm it is referred

to the back, often in the interscapular region or to the base of the neck depending on the level at which the growth lies

The second type of pain is constant, dull and boring and is felt principally in the back at a level slightly higher than that of the growth itself. This type is caused either by periesophagitis and mediastinitis due to the spread of infection from an ulcerated tumor or by local mediastinal involvement from an invasive carcinoma. Sometimes it is caused by spinal metastases. It usually denotes inoperability of the growth for the reasons given.

OBJECTIVE SIGNS In the early or even fairly well advanced stage of the disease the appearance of the patient is usually normal. In the stage of marked obstruction and ultimate metastases or complications, emaciation, asthenia and a yellowish discoloration of the skin may be noted.

Palpation of the neck may reveal the presence of a large growth in the cervical segment or of metastatic lymph node involvement from carcinoma in that or the superior mediastinal segment. Metastases to the neck from a tumor in the middle or lower thoracic segments are not frequent.

Peroral digital exploration of the mouth of the esophagus may reveal the presence of a tumor in that region. Percussion and auscultation are of little value except to give evidence of pleural or pulmonary complications.

ROENTGEN EXAMINATION in this stage of the disease shows clear cut deformity and irregularity of the contour of the mucous membrane with more or less protrusion into the lumen. The irregularity may cause filling defects seen in the barium column. These shadows present varied contours, often dentate or punched out in appearance. They remain constant during the presence of the barium in the esophagus (Fig. 362). Sometimes there are additional shadows above such a tumor due to the presence of particles of food.

If the tumor has not surrounded the esophagus the lumen may enlarge so much that attention may not be drawn to the fact that there is loss of elasticity of the esophageal wall. If on the contrary the tumor is annular, the passage presents a more or less characteristic narrowing, the upper limits of which often show an irregularity corresponding to the nodularities of the margin of the growth (Fig. 363). Sometimes there is a shelf-like appearance from the center of which a small trickle of barium descends through the much constricted lumen (Fig. 364).

With a small amount of barium the contrast liquid shows a disappearance of the mucosal folds and in their place irregularities and abnormal patterns (Fig. 365). Sometimes with an ulcerated growth a segmental enlargement of the lumen appears particularly in the recumbent posture and with only a little barium.

The opening through the tumor is usually eccentric, as would be expected with a growth which tends to develop irregularly. In rare instances of symmetrical annular development it is difficult to differentiate radiologically between a malignant lesion and a cicatricial stenosis. Here the history and other clinical manifestations are of great importance.

Proximal to the tumor the esophagus appears more or less dilated according to the duration and degree of the obstruction. Peristaltic activity in this segment is active or even exaggerated when the duration is short. Sometimes there is



FIGURE 362 Esophagogram showing a large filling defect with dentate contour indicative of a large irregular carcinoma (1) mediastinum invaded (2) (inoperable case)

an admixture of normal peristaltic and abnormal antiperistaltic contractions. In cases of long standing in which the stenosis is almost complete, the esophagus has lost its tonicity and its motility is almost completely lacking. One may just be able to discern an occasional undulation analogous to those seen on the greater curvature of an atonic stomach.

The inferior margin and therefore the longitudinal extent of the growth along the esophagus is difficult to define. This may be accomplished in some instances, however, by examining the patient in the recumbent position and encouraging by pressure or otherwise the reflux of barium into the lower esophagus from the stomach. The use of the Trendelenburg position may be helpful.

ESOPHAGOSCOPY. The findings vary. In case of infiltration a localized rigidity with loss of flexibility of the esophageal wall is noticed. This may be appreciated by palpation with the end of the tube although it cannot be seen. It is noticed also that the tube cannot be passed through this region, or at least

FIGURE 363 Esophagogram showing an annular carcinoma with partial obstruction (1) level of the aortic arch (?)



FIGURE 364 Esophagogram of an annular carcinoma with almost complete obstruction and marked proximal dilatation





FIGURE 365 Esophagogram showing a large irregular filling defect indicative of a large fungating type carcinoma. Bracket shows the longitudinal extent of the tumor.

it passes only with difficulty. There is also always evidence of esophagitis. The mucosa is fragile and may bleed easily when the submucosal infiltration is extensive enough to make it stand out.

A clearly defined ulcer is unusual, whereas the fungating type of tumor is frequent. The projections of such a tumor, which bleed easily, appear freely in the lumen (Fig. 366). Sometimes the growth is obscured by inflammatory tissue, a fact which explains the difficulty of obtaining a biopsy specimen and the necessity for several successive attempts in some instances.

Esophagoscopic views of various types of carcinoma may be seen by consulting Plates II and III.

Esophagoscopic biopsy is easy with fungating tumors, less so with ulcerating lesions. With infiltrating scirrhous tumors it is difficult to obtain a sufficiently deep bite with the biopsy forceps. When the report from the laboratory comes back negative or chronic inflammation, one must not conclude that the lesion is benign, for the bite is often too small or it was taken from the wrong place. The biopsy should be repeated unless there is little doubt. In the ulcerative type, however, it is better to give up the idea than to produce a perforation. In such a case tissue may possibly be obtained by curettage or wiping with gauze.

Better still cytological studies may be made of esophageal washings. In fact, the percentage of failures is lower with the latter method than with actual tissue biopsy through the esophagoscope.

In spite of all its advantages it must be admitted that esophagoscopy in carcinoma of the esophagus is not without its dangers. Deaths have been reported from opening the pericardium, from perforation of the air passageway, from perforation of a phlegmon, and from other causes.

The Terminal Stage

The terminal stage, which comes on rapidly at about the sixth to the eighth month from the onset of symptoms, is marked by progressive cachexia. The patient grows thin rapidly, losing ground visibly from day to day. He suffers thirst and swallowing becomes more and more difficult with attendant pain and regurgitation. Fistulae may develop. Recurrent nerve paralysis (usually the left) and sometimes respiratory difficulty are common. Metastases begin to give evidence of their presence, their manifestations depending upon the organ or tissue involved.

In this phase of the disease death often intervenes rapidly from acute pulmonary complications secondary to esophago-respiratory tract fistulae or from purulent empyema, pericarditis, or mediastinitis.

ROENTGEN EXAMINATION should be undertaken with caution because of the possible presence of a fistula into the air passages which might become inundated with barium mixture if much is ingested at once. The findings have been described above.

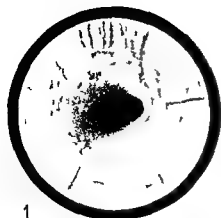
ESOPHAGOSCOPY at this stage should not be attempted save in unusual circumstances to establish a diagnosis in the occasional doubtful case. In this advanced stage there is danger of producing a fistula or of enlarging the opening of one already existing but latent. Sometimes there is danger of opening a large vessel.

Definite contraindications are the presence of fever, an esophago-tracheal or bronchial fistula, an aneurysm of the aorta or evidences of localized infection. The same is true also of pulmonary complications, cardiac decompensation, pericarditis, advanced tuberculosis and recurrent nerve paralysis.

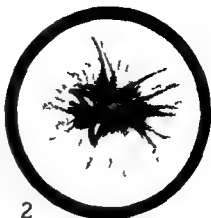
When done with caution the examination usually shows a growth with a cauliflower appearance or one which is ulcerated and has a tendency to bleed easily. The esophageal lumen is extremely narrow.

FIGURE 366 Esophagogoscopic view of a fungating polypoid carcinoma of the esophagus

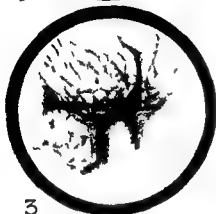




1



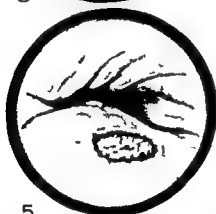
2



3



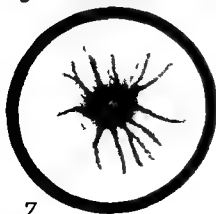
4



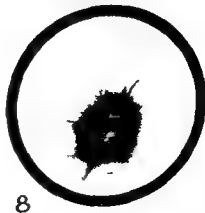
5



6



7



8

Special Considerations

Variations of the clinical manifestations of carcinoma of the esophagus may be grouped as follows

1 **ABNORMALITIES OF CLINICAL COURSE** A certain number of carcinomata arising in the lower portion of the esophagus begin with purely gastric symptoms with little or no dysphagia, and often with early metastases to regional lymph nodes. In these the diagnosis may be long delayed because of the lack of typical symptoms and the difficulties of making a reliable roentgen examination of the region of the cardia

Another peculiarity of onset is its rapidity in some instances the dysphagia appearing suddenly without premonitory signs. The first attack in such a case is usually precipitated by swallowing poorly masticated solid food or more rarely cold liquids. Sometimes a foreign body like a prune or peach pit or a morsel of meat provokes a sudden obstruction at the site of a small carcinoma and esophagoscopic intervention becomes necessary.

A further anomaly of onset is the relatively frequent occurrence of a latent type which gives rise only to an anemia. These do not cause dysphagia and may not be diagnosed except by esophagoscopy.

Occasionally the first sign of the presence of a carcinoma of the esophagus is hoarseness due to paralysis of one of the vocal cords from invasion and destruction of one of the recurrent laryngeal nerves, usually the left.

In other patients the first sign of a high-lying carcinoma of the esophagus is the appearance of a large mass of lymph node metastases in the neck.

In rare instances the initial episode may be the sudden appearance of hemiplegia or other evidences of intracranial metastases. An occasional case in which extensive liver metastases become manifest before the primary esophageal tumor is discovered has been noted.

Severe pain may be lacking in as many as 60 per cent of the patients. Dysphagia in 20 per cent.

Although excessive salivation may be absent, it is sometimes much exaggerated.

Variations in the duration of the disease are wide. The acute form observed usually in young subjects is very rare. In these the evolution is rapid, lasting only from one to four months. This is seen usually in patients under 45 years of age.

In aged persons the disease may pursue a languid course with gradual development of moderate degrees of weight loss and a slight amount of dysphagia. In these the total duration may be as long as one and one-half to two years.

In other instances after a period of intense dysphagia the patient may suddenly experience striking relief even to the point where he can consume solid food again. Sometimes it becomes difficult to convince the patient that he still

PLATE III THE PATHOLOGICAL ESOPHAGUS (ENDOSCOPIC VIEWS)

1 Cicatricial stenosis with esophagus 2 varices of the esophagus 3 carcinoma of the esophagus (proliferating type) 4 carcinoma of the esophagus (submucosal type) 5 carcinoma of the esophagus (ulcerated type) 6 spasm of the mouth of the esophagus 7 spasm of the phrenocardiac region 8 esophageal polyp (biopsy is indispensable)

has something wrong until the dysphagia, after a period of a few days to a week or more, begins to recur. This phenomenon is caused by the sloughing of the necrotic central part of the tumor which automatically enlarges the lumen once the necrotic tissue has become separated from the remaining body of the growth (Fig. 361).

Sometimes after the necessity for swallowing has been supplanted by the institution of a program of gastrostomy, feeding deglutition may improve as a result of the subsidence of secondary inflammatory swelling and esophagospasm.

With carcinomata which exhibit a striking degree of dilatation, it is necessary to distinguish between that produced by the slow development of a constricting scirrhus growth and a case of mega-esophagus in which a carcinoma has developed. In the latter instance the malignant degeneration occurs late, many years after the onset of the achalasia. Its development is essentially silent until loss of weight, emaciation, cachexia, a more or less painful dysphagia and often intercurrent thoracic complications like respiratory fistulae or lung abscess make known the presence of a carcinoma which until then has caused no symptoms.

The diagnosis is difficult in a case of mega esophagus even with the use of fluoroscopy, roentgen films, and sometimes esophagoscopy. If the esophagus is thoroughly emptied before the endoscopic examination, however, the tumor can usually be seen. The trouble lies in the fact that, because of the lack of symptoms in such a dilated esophagus, the disease is usually far advanced when it is first discovered.

Sometimes such an enormously dilated esophagus can be more successfully examined with a Schindler gastroscope than with the esophagoscope.

In an occasional patient the first evidence of the presence of a malignant neoplasm may be the finding of positive cells in washings obtained from the esophagus. This circumstance must be very unusual unless the policy is adopted of performing such an examination in all patients who have achalasia. The relatively high incidence of carcinoma as a complication of this disease makes this practice advisable.

2. VARIATIONS DEPENDING ON THE LOCATION AND ANATOMICAL TYPE OF THE TUMOR. *Carcinoma of the Mouth and Cervical Segment of the Esophagus.* It is well known that the former predominates in women and that it bears an etiological relation to the pre-existence of the keratotic changes in the mucous membranes which are characteristic of the Plummer-Vinson syndrome (see Chapter 13). Pharyngeal and laryngeal symptoms are pronounced in this type of the disease with bouts of coughing, dysphagia, difficult deglutition, and spasm. Complications of the air passages occur early and are frequent. The diagnosis is always difficult. In the presence of persistent accumulation of saliva within the pyriform sinuses, a roentgen examination of the upper esophagus and pharynx is indispensable (Figs. 367 and 368) and direct inspection using a laryngeal speculum should be employed to enable the performance of a biopsy.

Carcinoma of the cervical segment likewise is more common in women than in men. No reason for this is apparent. The symptoms are vague at the onset with burning sensations, pharyngeal tenesmus, the feeling as though there were a foreign body present, and often other less well defined sensations or

paresthesiae' which vary with the patient. The most reliable symptom is a striking dysphagia which appears principally with meals. Usually in true paresis the difficulty is alleviated by swallowing food or taking a mouthful of water.

As mentioned above, carcinomata of the lower or juxtacardiac segment often present a special aspect with symptoms more gastric than esophageal in character. Sometimes the gastric symptoms are striking with vomiting, hematemesis and weight loss, absence of dysphagia and a negative roentgen examination so far as the esophagus is concerned. In other patients the gastric symptoms may be more like those of an ulcer, with exaggerated epigastric pain and hyperchlorhydria although characteristically the free acid level is low. Dysphagia ultimately assumes an important role in the symptomatology.

The diagnosis may be particularly difficult even with the use of the esophagoscope. In many instances a carcinoma in this location actually arises in the stomach with secondary involvement of the esophagus by direct extension. The majority of these tumors are histologically adenocarcinomata indicating their gastric origin though in some instances a growth of this type may arise from intrinsic esophageal glands. Often the tumor has a tendency to infiltrate by way of the submucosal layer. When viewed through the esophagoscope the mucosa appears heaped up by the presence of the tumor beneath and the esophageal wall appears rigid and immobile (Plate II, No. 9).

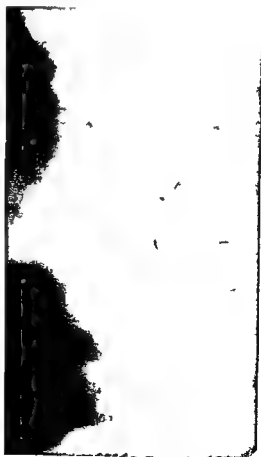


FIGURE 367. Roentgenogram of carcinoma at the mouth of the esophagus on the left lateral wall. No obstruction.



FIGURE 368 Carcinoma of the mouth of the esophagus showing stasis in the valleculae and pyriform sinuses

Complications

The pathological anatomy of the various complications of carcinoma of the esophagus has already been described. The clinical manifestations of the most important of these require special consideration.

Complications Involving the Esophagus and Its Surrounding Tissues

Temporary, complete obstruction of the stenotic lumen at the site of the tumor, caused by the impaction of a piece of meat, a fragment of bone, a fruit pit (cherry, orange, plum, etc.), is a common occurrence. The accident makes further deglutition impossible and if the foreign body is not dislodged by vomiting or by its passage into the stomach, it may be necessary to remove it through the esophagoscope.

Esophagitis superimposed on carcinoma of the esophagus is manifested by more or less acute pain, elevation of temperature, and an exacerbation of dysphagia. It occurs as a consequence of stagnation of food or of the lodgement of a foreign body in or above the obstructing tumor. The inflammation may spread from the esophageal walls to cause mediastinitis or peritonitis.

Mediastinitis is rather frequent in the advanced stage of the disease when

a large ulcerating tumor becomes infected. It may end in a phlegmonous or purulent (usually putrid) abscess. Another cause, of course, is a perforation resulting from imprudent or unskillful endoscopic manipulations.

✓ *Peritonitis* is usually the result of a perforation at the level of or just below the diaphragm. It may be manifested by the presence of a subphrenic abscess which may not be clinically obvious at first, or more often by rather rapid death from overwhelming widespread infection throughout the abdominal cavity. It is often instrumental in origin.

• *Adenopathy* involving the regional lymph nodes consists usually of metastatic involvement but in some instances the process is purely inflammatory and secondary to the complicating infection of the growth. Sometimes both types of nodal pathology are present in the same area.

Lymph nodes which are infiltrated with metastatic carcinoma may give rise to paralysis of the recurrent phrenic, or vagus nerves because of direct tumor invasion of the nerve trunk. The left recurrent nerve is more frequently involved than the right. Clinical evidence of involvement of any of these nerves lies in the presence of spasms of coughing, of hiccoughs, roentgenological observation of a paralyzed diaphragm, and visualization of the vocal cords by laryngoscopy.

✓ *Laryngeal and tracheal complications* are important. They are particularly frequent in carcinoma of the cervical segment which is so relatively common in women. Sometimes the larynx becomes involved by direct extension from the hypopharynx. Primary carcinoma of the larynx may be an occasional associated lesion. Rarely the laryngeal involvement may be the result of hematogenous metastasis from an esophageal carcinoma in the lower esophagus. Invasion of the recurrent nerves and of the trachea gives rise to a barking cough or sometimes a combination of a bitonal voice and the cough due to compression. Acute episodes may require a tracheostomy to relieve obstruction, but this is frequently useless. If the tracheal involvement is high the stenosis may be overcome by inserting an extra long cannula, but this is an unusual situation.

Pulmonary infection rapidly supervenes with bronchopneumonia, pulmonary abscess or gangrene, often complicated by empyema. Bacteremia resulting from all this sepsis may be the terminal event.

• Roentgen studies of the chest may reveal the presence of pulmonary metastases.

✓ *Fistulae* may be of two clinical types. The latent form is one where an actual intercommunication between the esophagus and the trachea or bronchus is plugged by a nodule or outcropping of tumor or even by a foreign body such as a nut or seed or by the obliquity of the tract, which produces a valvular effect. In any such case instrumental manipulation such as the removal of tissue for a biopsy attempts to dilate the constricting tumor or the extraction of a foreign body may open up the fistula tract with disastrous results. A latent fistula often becomes obvious after sloughing of the tumor in response to roentgen therapy.

Fistulae with obvious signs are better known. They may cause symptoms even though the tract is small in diameter. The most important evidence for their presence is furnished by fluoroscopy and roentgen films. As mentioned before, however, a source of error exists when there is pharyngeal or laryngeal

paralysis, as a result of which small amounts of barium may spill into the air passages during attempts to swallow

The prognosis is grave and death follows rapidly. During the brief survival of the patient the fistula, once it has been formed, interferes enormously with eating and leads to the serious respiratory tract complications already mentioned. Rare exceptions where the patient has survived several months after the development of a fistula of this sort are on record.

Cardiovascular Complications

Compression of the superior vena cava by lymph node metastases in the superior mediastinum is manifested by the usual signs characteristic of superior mediastinal venous obstruction.

Venous thrombosis is more unusual. It gives rise to variable clinical manifestations.

Thrombophlebitis of the leg veins may occur as in any other debilitating illness.

Hemorrhage may occur, but rarely from the tumor itself except for slight blood loss manifested by positive chemical tests of the stools. Cataclysmic arterial bleeding is not unusual as the terminal event. It results usually from erosion of the aorta and gives rise to the sudden vomiting of large quantities of blood, ending in death from exsanguination. Instrumentation through the esophagoscope or the sloughing of a tumor induced by irradiation may be the inciting causes.

Pericardial involvement occurs late in the disease and is usually recognized only at autopsy.

Rarely there may be metastases to the heart muscle from which disturbances in rhythm may result.

Complications Involving the Diaphragm

In cases of carcinoma of the lower esophagus the diaphragm is often invaded by direct extension of the tumor. Paralysis of the diaphragm from phrenic nerve involvement by way of lymph node metastases has already been mentioned.

Hiccoughing because of phrenic nerve irritation is more unusual.

Roentgen examination is important in the study of any of these complications.

Complications Involving the Nervous System

Complications involving the nervous system may be either central or peripheral. The former, due to cerebral or meningeal metastases, are most unusual, in contrast to carcinoma of the lung in which they are frequent. Involvement of the spinal cord or nerve roots is more common. It may be due to metastases, but more often it is caused by direct extension from mediastinal metastases or vertebral involvement. The symptoms of either of these conditions depend naturally upon the location of the nerve lesion. Sometimes the brachial plexus is involved with atrophy, pain, paresthesiae, or anesthesia of the arm or hand.

Peripheral nerve lesions, especially of the recurrent laryngeal nerves are the most frequent of all. Lesions of the vagus and phrenic nerves observed in carcinomata of the tracheo-aortic region are manifested particularly by attacks of dyspnea and substernal distress. These have been alluded to.

Paralysis of the superior laryngeal nerve causes anesthesia of the laryngeal mucosa and facilitates the escape of food or of barium mixture into the larynx during deglutition.

The sympathetic trunk is frequently involved. Contraction of the pupil, enophthalmos, narrowing of the palpebral fissure, warmth and dryness of the skin of the face are the classical signs when the cervical sympathetic chain is involved (Bernard-Horner's syndrome).

RECURRENT LARYNGEAL NERVE INVOLVEMENT is of special importance, often bringing about an acceleration of the course of the disease. For obvious anatomical reasons the left nerve is more frequently affected than the right. Carcinomata located in the cervical or upper mediastinal segments may cause the difficulty by direct extension. Invasion of the nerve by metastases in adjacent lymph nodes may cause it, no matter where the primary tumor may be.

The classic clinical sign is the so-called "bilateral voice." The cord, generally at first in the median or juxtamedian position with immobility of the arytenoid ultimately takes the lateral or cadaveric position. The opposite cord, however, can during phonation compensate to a certain degree for the inadequacy of the paralyzed cord. Variations occur, the interpretation of which is sometimes difficult.

In the presence of this type of laryngeal paralysis (left cord), the diagnosis in the absence of other suggestive symptoms rests between esophageal carcinoma and aneurysm of the aortic arch. Roentgen examination in addition to the clinical history serves usually to differentiate the two.

BILATERAL RECURRENT NERVE PARALYSIS is much less frequent, occurring almost exclusively in cases of carcinoma in the cervical segment, usually with complicating lymph node metastases. Two clinical types may be distinguished, namely the *dyspneic* and the *aphonic*. The first is characterized by marked adduction of the cords, a raucous voice without true aphonia and intermittent dyspnea which finally becomes constant and requires a tracheostomy. The prognosis is grave from the first and the downhill course is rapid.

In the second there is more or less complete aphonia with a total lack of dyspnea. The course is gradual and attacks of suffocation do not occur. In these the paralysis takes place first on one side and then on the other with more marked abduction on one than the other.

Diagnosis

Symptomatic

Progressive dysphagia is the outstanding symptom. It is regrettable that the public and even many physicians are not sufficiently aware of this fact. Since the advent of surgical measures to eradicate the disease it is discouraging to be confronted by the knowledge that the diagnosis is often made too late for successful intervention to be carried out. Many have sought to establish early signs

of cancer of the esophagus, but until the day comes when the diagnosis can be made at the outset, before the appearance of dysphagia which is almost always too late a symptom, the statistics cannot be much improved. Every effort, therefore, must be made on the part of both the patient and the physician to recognize the lesion in its early stages.

The dysphagia which is the principal evidence of the presence of carcinoma is a constant symptom produced by the tumor itself, by superimposed esophagospasm, or by the rigidity of the wall of the organ at a limited point. It is especially frequent in the infiltrating type of tumor which obstructs the peristaltic waves. The same is true for the ulcerative form. It occurs less early in the vegetative type of growth which causes a more gradual obstruction of the lumen and interferes mechanically with the passage of food.

Any complaint of persistent dysphagia especially for solids, no matter how slight, is a cause for concern which demands immediate investigation. Much too often patients are seen who have been treated for months with various powders or have been advised to take a protracted vacation because of assumed neurotic tendencies without any examination having been made. The opportunity for successful surgical intervention is thereby lost. The inexorable end may be more or less retarded, though often it may be accelerated by the measures called 'palliative' or 'humane' which only prevent the patient from dying of starvation.

With carcinomata at the mouth of the esophagus or in the cervical segment, dysphagia is exceptionally troublesome and is painful. The patient, almost always a woman, complains of burning or prickling sensations or of a feeling as though there were a foreign body present. These sensations are described in various ways. In the little-understood condition spoken of as "pharyngeal paresthesia" the pain stops during a meal and the patient stresses clearly this moment of relief. In this situation particularly there is a certain obsession and if the patient forgets about it for a moment, when questioned later he always insists with greater emphasis that the trouble is still present. Contrariwise, in the dysphagia which has an organic basis the difficulty is maximal during a meal and the dysphagia is permanent, painful, and unremitting.

Patients with a functional or neurotic complaint are often fearful of cancer and consult the physician with that in mind. They are often dissuaded with difficulty. The patient with an actual malignant growth, on the other hand, often ignores the danger and it may take some effort to convince him.

In the presence of a lasting dysphagia the physician must not rely upon the clinical signs and symptoms alone, which may be deceiving. His role is to advise the patient without delay, for it cannot be repeated too often that dysphagia frequently indicates the presence of a lesion which is far advanced. One should never wait for the appearance of the classical signs described in the textbooks. These are always late signs.

This is true, for example, of *salorrlia*. It is better to discover a persistent pooling of saliva in the pyriform sinuses with the laryngeal mirror than to wait for the patient to develop drooling from the mouth.

The same situation applies to the occurrence of *pain*. A patient with an

esophageal carcinoma who experiences persistent pain especially in the back, has a far advanced lesion which is almost invariably inoperable

There is no point either in stressing the sign too often described of a *foul breath*. This fetidness indicates a necrotic cauliflower type lesion of the esophagus or a deep infected ulcer. As mentioned elsewhere the esophagus is a particularly contaminated organ and infection is usually superimposed upon any extensive neoplasm.

It is always wrong, as is done in the textbooks, to attempt to distinguish between the initial phase of onset and the period of full development of a cancer and to mix the symptoms. Practically speaking the treatment of malignant neoplasms can be applied successfully only to those which are limited, circumscribed and removable, those which the surgeon can extirpate with a wide margin of surrounding normal tissues. Once the lesion has penetrated the wall of a fragile, poorly defended organ (a frequent situation with cancer of the esophagus) and has invaded the periesophageal tissues the percentage of good results falls noticeably.

Roentgen Examination

Roentgen examination, which must be carried out in any patient with a complaint of dysphagia is of enormous value in making the diagnosis. In fact, the number of cases of carcinoma of the esophagus in which further esophagoscopy confirmation is required is small.

The fluoroscope serves to localize the lesion and the films taken in various projections give an opportunity to study its configuration. Localized (spot) films are helpful but it is important for the orientation of the surgeon who is to carry out treatment to have the benefit of esophagograms which show the entire organ well.

Esophagoscopy Examination

Endoscopic examination is of great help but, because of the occurrence of serious complications in the hands of some operators there is a widespread opinion among physicians generally that the maneuver is dangerous and painful. This idea must be vigorously combatted because it is incorrect and because esophagoscopy alone may be the only means of making a diagnosis of a neoplasm in its early stages when the taking of a biopsy is so important to prove the nature of the lesion.

In the early lesion, excepting those which are submucosal at the time is biopsy is virtually always positive. Many times when the neoplasm is obvious on the roentgen film however the biopsy is not always conclusive. This apparent paradox is due to the fact that the more advanced lesion is obscured by the edematous inflammatory reaction of a complicating esophagitis. In these it is possible to reach the neoplastic tissue only by taking specimens from various levels throughout the constricted area.

Without a positive biopsy there is some risk either of exploring a patient who has nothing but a localized area of spasm possibly a reflex tonic contraction secondary to a gastric ulcer or other process in the upper alimentary canal, or of removing an inflammatory stricture which might have been treated otherwise.

In practice it is necessary to use clinical judgment. The conduct of the investigation must depend upon the knowledge and experience of the physician and surgeon in charge as well as the availability of an endoscopist (often the surgeon) who can carry out the procedure with safety. In many instances, therefore, it is justifiable when the roentgen appearance is unequivocal to proceed with surgery without an esophagoscopy. In the occasional case in which a nonobstructing lesion which might be malignant is found, or there is reason to believe that a partially obstructing lesion may not be malignant and can therefore be treated by measures other than surgery, it is imperative to insist upon the performance of an esophagoscopy. The examination is not necessary as a routine in all patients.

Cytological Examination

It should be pointed out in further substantiation of this viewpoint that *cytological studies of secretions* obtained from the esophagus proximal to a suspicious lesion have proved to be exceedingly accurate when made by an experienced person. In a large series of patients with proven carcinoma of the esophagus the examination was positive in 98 per cent of the cases. In some of these patients, tissue biopsies obtained at esophagoscopy were negative because of the technical difficulties inherent in the method. Conversely, in a series of patients with esophageal lesions presumed to be nonmalignant (achalasia, stricture, etc.) who were examined in this way as a control, an unsuspected carcinoma was occasionally found by the cytological technique.

In the present day, therefore, it is entirely proper to proceed with treatment in any patient with a suggestive roentgen appearance, especially when confirmation by adequate cytological studies of material obtained from the esophagus has been made. Esophagoscopy may then be reserved for doubtful cases, especially with an undiagnosed small lesion and in patients who might be spared a thoracotomy if the presence of a carcinoma can be definitely excluded.

Differential Diagnosis

In the differential diagnosis of carcinoma of the esophagus it is necessary to consider, first, the conditions which cause extrinsic pressure on or involvement of the esophagus, and secondly, nonmalignant intrinsic lesions.

Extrinsic Lesions

In the cervical area, extrinsic involvement or compression may be due to brachial cleft carcinoma or those arising in the larynx, low pharynx, or thyroid, as well as lymph node metastases from any of these lesions. External compression by a constantly filled diverticulum may be encountered as a source of diagnostic confusion.

In the thorax in addition to filled diverticula one must consider pressure by benign tumors, simple or malignant endothoracic goiters, lymphadenopathy, posterior mediastinal tumors such as lymphomata, inflammatory lesions such as mediastinal abscesses, tuberculous lymph nodes, cardiac enlargement, and

aneurysms of the aorta which must be recognized before any attempt at endoscopic examination is made

In the differentiation of *aortic aneurysm* dysphagia, which may be progressive in carcinoma is irregular in aneurysm. Recurrent nerve paralysis is more unusual (7 to 10 per cent of cases) in carcinoma than in aneurysm (30 per cent). Left nerve paralysis is common to both conditions, but right recurrent nerve involvement is less exceptional in carcinoma. Bilateral paralysis is seen more often in carcinoma than in aneurysm, where it is rare. Along with these signs common to both there are certain signs distinctive of aneurysm like the Oliver sign and obliteration of the radial pulse. These may also be seen in carcinoma, though rarely.

In doubtful cases the differentiation is made by fluoroscopic examination and the study of roentgen films the distinguishing details of which need not be mentioned here. It may sometimes be helpful to employ angiocardiology. Esophagoscopy is contraindicated whenever there is a reasonable prospect that the esophageal disturbance is caused by the pressure of an aneurysm. There is too much danger of breaking into the aorta with the obvious fatal outcome from hemorrhage.

In the abdominal segment aneurysm of the aorta in that region may cause external pressure. Metastases from carcinoma of other organs to the paracardial group of lymph nodes may also be a source of confusion.

Intrinsic Nonmalignant Lesions of the Esophagus

Impacted *foreign bodies* previously unsuspected but discovered by roentgen studies may simulate a carcinoma, especially in the mentally deranged who are incapable of giving an accurate history. Traumatized areas may also present a suggestive appearance.

Inflammatory lesions are rarely confused with carcinoma in their acute phase. It is unusual also for *congenital stenoses* of the esophagus to become manifest so late in life as to bring up the question of malignant tumor. Such cases are on record, however, but the roentgenological and esophagoscopy examinations as well as a history of more or less troublesome dysphagia from childhood permit the differentiation.

The diagnosis is more difficult in the case of benign so-called *peptic ulceration* of the esophagus.

The same is true of *tuberculosis* and *syphilis* except that the extreme rarity of these conditions makes their diagnosis unlikely.

Lymphogranulomatosis and *actinomycosis* are two extremely rare conditions which may simulate carcinoma of the esophagus.

Esophagospasm when tenacious and progressive may lead to inflammation of the esophageal wall and proximal dilatation thus provoking a degree of dysphagia which simulates that of carcinoma. It is principally the spasm which occurs in the lower esophagus which may lead to an erroneous diagnosis. Even with the benefit of esophagoscopy the difficulty is sometimes considerable when it comes to differentiating chronic spasm ending in stenosis from interstitial carcinoma especially in the region of the cardia. Even a biopsy does not always



FIGURE 369 *A* Pedunculated polypoid carcinoma of the esophagus 1, Tumor in the grasp of a forceps 2 esophagus showing pedicle of tumor covered with normal appearing mucosa *B*, Esophagogram of the patient before operation showing the peculiar appearance of the tumor in the lower esophagus Man aged 76

give results in lesions of the lower esophagus because the bite is often not deep enough

Differentiation between carcinoma and *benign tumors*, excepting in the case of unusually small tumors, is not ordinarily difficult. Ninety per cent of all esophageal tumors are actually carcinomata. Sarcomata are rare. On the other hand, from time to time more or less serious stenoses due to *benign tumors*, especially papillomata, are encountered. The differentiation between these and the papillomatous form of carcinoma is difficult, especially after a negative biopsy which may have come from the superficial part of the tumor. Cysts, adenomata, fibromata, lipomata, and myomata can simulate a very slowly developing submucosal carcinoma. The roentgen appearance of these benign intramural tumors is so characteristic that confusion rarely arises.

Polypoid tumors are usually benign, but an occasional polypoid carcinoma may be seen (Fig. 369).

The coexistence of both malignant and benign tumors in the same esophagus has already been mentioned.

CHAPTER 28

Surgical Treatment of Carcinoma of the Esophagus

THE PRINCIPAL objective in the treatment of carcinoma of the esophagus is the relief of the dysphagia which renders these patients so miserable. By far the best means of accomplishing this objective is the ablation of the tumor, provided a satisfactory functional substitute for the segment removed can be provided. In addition, whenever, as a result of our efforts to relieve the patient of his misery by excising the growth, we are favored by a prolongation of life to five or more years, at which time it is reasonable to believe that a cure has been effected, it must be acknowledged that an additional fortuitous benefit has been obtained. It is striking to observe how frequently this comes about in the course of operating upon a large number of patients with this disease.

As a question of policy, therefore, in order to secure the maximum degree of comfort for the largest number of patients with this disease, it is essential to remove the growth in every patient whose condition is satisfactory enough to permit the performance of the operation with a reasonable risk. This means, however, that in a large percentage of the patients the operation will be purely palliative because of the obvious necessity for leaving behind inaccessible distant metastases or invaded lymph nodes which lie beyond the scope of the resection. As will be seen later in evaluating the results of treatment, it is necessary to make a separate category of patients in whom this situation exists in contradistinction to the much smaller group in which the growth is localized and the lymph nodes are free from metastases or only a few of the nodes are involved.

In every case, however, the effort should be made to approximate as closely as possible the ideal operation for the removal of a carcinoma of any organ, namely, complete extirpation of the primary lesion including a wide margin of surrounding normal tissues and an en bloc removal along with the specimen of as many as possible of the regional lymph nodes in which metastases so frequently occur.

The relative success with which this ideal may be realized in treating carcinoma of the esophagus depends upon the anatomical relations of the segment in which the carcinoma lies. The prospects are for this reason least favorable in the cervical segment and most favorable in the lower or juxtacardiac area, with all gradations from poor to more favorable in the direction of above downward in the areas between.

In the cervical segment, although restoration of continuity could be made by using the stomach, the jejunum, or preferably the colon, it is practical only to use a segment of skin of the neck to restore continuity. The difficulties of removing a wide enough group of lymph nodes to which the tumor may have spread make surgical excision of carcinomata in this area inadvisable except in the most favorable early cases. This is because, in order to remove all the lymph nodes which might be involved, it is necessary to perform a bilateral radical cervical lymph node dissection as well as to remove all the nodes in the mediastinum, a procedure too formidable to be carried out in the average patient.

The result is that surgical excision for carcinomata of the retrocricoid and cervical segments must be confined to the treatment of small, presumably early tumors which do not show evidence of lymph node involvement. This means that in only about 30 per cent of the patients with carcinoma of the cervical esophagus is surgical treatment advisable.

In the fortunately small number of patients whose carcinoma is in the superior mediastinal segment (between the base of the neck and the aortic arch), a similar situation prevails. In this area also, for anatomical reasons, it is not possible to perform what might be called 'a good cancer operation'. Metastases to the cervical nodes are relatively frequent and those in the superior mediastinum cannot be removed widely enough to provide assurance that much of the involved tissue has been excised if the tumor has spread beyond the primary site. In this region, also, not over one-third of the tumors are resectable in the ordinary sense of the term. From this region down, however, the prospects of a reasonably satisfactory eradication improve steadily the lower the tumor lies.

With tumors arising in the midmediastinal segment behind and just below the arch of the aorta there is a definitely better prospect of being able to remove both the local tumor and some of the regional lymph nodes of the subcarinal and periesophageal groups, as well as those below the diaphragm. These comprise the paracardial and left gastric groups in which metastases may occur with increasing frequency the lower the tumor lies in the mediastinal portion of the organ. In the midesophagus it is possible to resect the growth in approximately 69 per cent of the patients who are operated upon. The slightly greater prospect of removing all the nodal metastases in these patients, also, is reflected in the improved prognosis in terms of five year survival among those with the more favorable tumors (see below, page 570).

The closest approximation to the ideal cancer operation in carcinoma of the esophagus is reached in the case of tumors in the lower segment and at the cardia. With these it is possible to excise a long segment of normal esophagus proximal to the tumor, a large portion of uninvolved stomach below, and almost all of the periesophageal nodes in the mediastinum below the level of the aortic arch along with the vastly important paracardial and left gastric groups as well

the splenic and pancreatic nodes in some instances, provided that a splenectomy and distal partial pancreatectomy are included as a part of the procedure. The resectability among patients operated upon for carcinomata in this area is close to 85 per cent. Furthermore, because of the more widespread removal of the disease made possible by the location of the growth, the end results, as will be seen below, are comparable to those for carcinoma in other organs where surgery has become the accepted treatment (page 571).

Operations for Carcinoma of the Thoracic Segments

TECHNIQUE

The technical details of the operation vary depending upon the segment in which the carcinoma lies. The procedures which are adapted to the problem as it arises in each of the several segments are presented in order, beginning at the lower end.

Operation for Carcinoma of the Lower Esophagus and Cardia. Partial Gastrectomy and Esophagectomy with Low Intrathoracic Esophagogastric Anastomosis

The usual endotracheal inhalation anesthesia is used. The patient is placed on his right side with the left side arched upward slightly (Fig. 370). A standard thoracotomy incision is made using the rib resection technique. Modifications of the incision which may be required under certain conditions will be mentioned subsequently. The lung is palpated for metastases and the lower portions of the esophagus and mediastinum are explored. The diaphragm is then opened by a small incision and the abdomen is explored. The liver, stomach, spleen and regional lymph nodes are observed. If the growth is resectable, the division of the diaphragm is completed through the esophageal hiatus and the dissection is begun.

The left phrenic nerve is crushed to quiet the left half of the diaphragm.

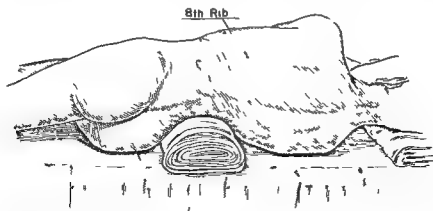


FIGURE 370 Position of the patient for thoracic esophagectomy showing the direction of the incision. Note: If a supra-aortic anastomosis must be performed, the incision is subsequently enlarged upward posteriorly between the scapula and the spine.

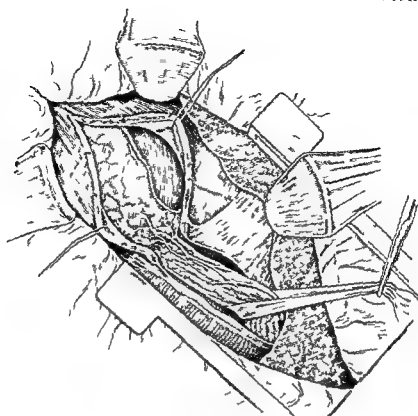


FIGURE 371 Mobilization of the lower esophagus and cardia. Diaphragm incised completely through the esophageal hiatus.

and to prevent diaphragmatic pull upon the stomach during the postoperative period.

TECHNIQUE OF DISSECTION Through a longitudinal incision in the mediastinal pleura posterior to the pulmonary ligament, the lower end of the esophagus is freed (Fig. 371). The right and left vagus nerves are severed at a point just above the probable level of division of the esophagus. Small vessels which accompany these nerves must be ligated. The dissection is then carried downward, dividing the attachments behind the cardia. In this region several vessels which anastomose with the lower esophageal, left phrenic, left gastric, and left superior suprarenal arteries are found. These must be cut and ligated. In the case of a large, locally invasive growth it may be necessary to remove a rim of the diaphragm along with the tumor. The gastrosplenic ligament is incised. This gives access to the lesser omental cavity. If the growth has begun to adhere to the splenic artery or has invaded the tail of the pancreas, the dissection is planned to provide for the removal of the spleen and often the distal portion of the pancreas. If the growth is not thus invasive, the spleen and pancreas are not removed. The left gastro-epiploic vessels and the vasa brevia between the spleen and the stomach are severed and tied. The gastrosplenic ligament is divided as far as the level of the pylorus, taking care to avoid injury to the anastomotic arches of the right gastro-epiploic vessels which lie close to the greater curvature. The gastrohepatic ligament is divided almost to the level of the pylorus, preserving the right gastric vessels which course along the lesser curvature. In

the proximal portion of the gastrohepatic ligament an artery (accessory left hepatic) is frequently encountered which arises from an ascending branch of the left gastric artery and extends within the ligament to the porta of the liver. This must be divided as a part of the mobilization of the stomach.

The last attachment which must be freed in order to complete the mobilization of the upper portion of the stomach is in the region of the left gastric artery. This vessel should be divided close to its origin from the celiac axis in order to make it possible to remove the large group of lymph nodes which surround its branches as they approach the lesser curvature of the stomach. The left gastric vein and the celiac branch of the right vagus nerve must also be severed (Fig. 372).

A second pair of gauze pads is used to protect the field of operation in anticipation of cutting across the stomach. Two large curved gastric clamps are applied to the stomach as far as possible below the lower extent of the growth. The clamps should be placed so that as long a portion of the greater curvature as is compatible with an adequate excision of the diseased area remains. The stomach is then cut between the clamps with a knife. A pad of gauze is tied over the proximal cut end and the tumor-bearing segment is turned back over the posterior angle of the incision. Division of the esophagus, however, is postponed until the posterior layers of the anastomosis have been completed. In this way the necessity for the use of a clamp on the proximal portion of the esophagus is avoided and soiling of the field is postponed until the anterior layers of the anastomosis are begun.

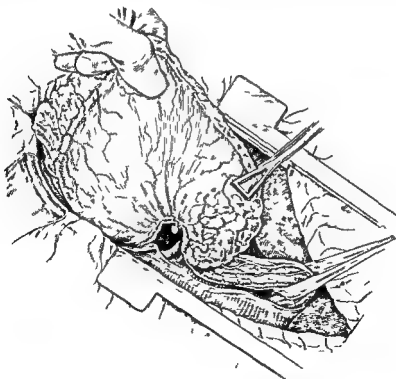


FIGURE 372 Mobilization of the lower esophagus and upper half of the stomach completed. Left gastric artery has been severed.

The distal cut edge is closed with two layers of fine catgut reinforced by an outer layer of interrupted Lembert sutures of silk. A convenient method of placing the catgut layers is to sew through and through below the distal clamp with a straight atraumatic needle, starting at the greater curvature end. If a clamp with fenestrated blades is available, the suture is passed through the blades. When this type of clamp is used, the application of the proximal clamp may be deferred until the through-and-through suture has been completed. The second clamp is applied and the stomach is cut between. The distal clamp can then be removed without spilling the gastric contents.

Using the same strand of catgut, the cut edges of the distal portion are sutured over and over from the lesser curvature and back to the starting point at the greater curvature. This part of the suture controls the bleeding which arises from the severed intramural gastric vessels. The closure of the end of the stomach is completed with two more layers of suture, the first consisting of a continuous inverting (Cushing right-angle) stitch of catgut and the second of interrupted Lembert sutures of fine silk.

After the end of the distal segment of the stomach has been closed, a circular incision is made through the serous and muscular coats of its anterior wall near the upper extremity, avoiding if possible any injury to the intramural vessels. This incision should be placed where no important branch of the gastroepiploic vessels may be encountered. Furthermore, it should not be made too close to the divided end of the stomach because of the danger of interference with the blood supply of the bridge of gastric wall which lies between. The diameter of this opening which is to be used for the anastomosis should be

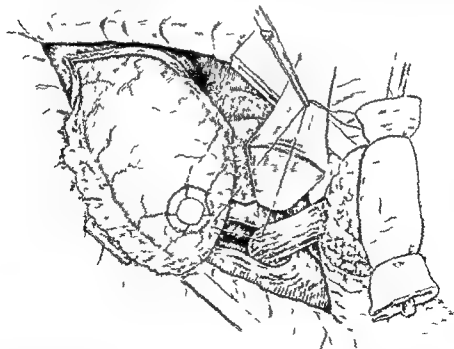


FIGURE 373 Start of the first layer of the posterior aspect of the anastomosis. A circular incision through the muscular coat of the stomach wall prepared for anastomosis. Vessels crossing the area to be excised have been ligated with suture ligatures.

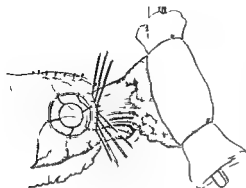


FIGURE 374 Outer layer of sutures of the posterior aspect completed ready for cutting saving the one on each end for traction

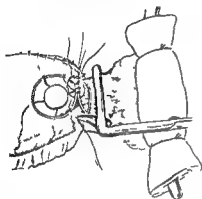


FIGURE 375 Edge to-edge approximation of the divided muscularis of the esophagus and the seromuscular coats of the stomach posterior aspect second layer

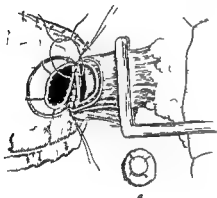


FIGURE 376 Edge to edge approximation of the posterior mucosal layer after excising the circular portion of gastric wall outlined by the incision (a)

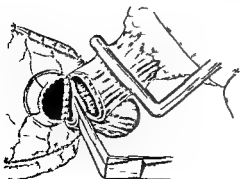


FIGURE 377 Excision of the esophagus cutting through all layers of the anterior wall using specially devised right angle scissors

FIGURE 378 Start of the mucosal layer of sutures on the anterior aspect of the anastomosis. Note: The sutures are inserted so that when tied the knots lie on the inside (intraluminal).

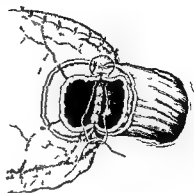


FIGURE 379 Second layer of the anterior aspect: esophageal muscle to seromuscular coat of stomach.

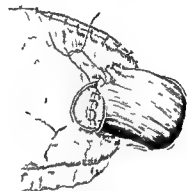


FIGURE 380 Outer layer of the anterior aspect. Note: These should be mattress sutures.



approximately equivalent to that of the esophagus when slightly distended. The final excision of this circular portion of stomach wall is postponed until after the first two layers of the posterior aspect of the anastomosis have been placed and tied. This is done to delay the soiling of the field with gastric contents until the last possible moment. After the circular incision has been made, a series of suture ligatures of very fine silk is applied to the small vessels which cross the area in the submucosal layer (Fig. 373). This step facilitates the completion of the anastomosis by diminishing the amount of bleeding which comes from the edges of the gastric wall after the circular piece has been removed.

The anastomosis is now begun with a layer of mattress sutures of fine (5-0) silk between the muscularis of the esophagus and the seromuscular coats of the stomach wall (Fig. 374). After the completion of this layer, a right angle clamp is applied to the esophagus just proximal to the upper reaches of the tumor. The muscle layer of the esophagus is next incised as far as, but not

through, the mucosal layer. This allows the placing of a second row of silk sutures to approximate the cut edges of the muscularis of both organs (Fig. 375). The posterior portion of the mucosa of the esophagus is then incised. A small opening is next made through the mucosa of the stomach and a curved aspirator is inserted to remove whatever liquid may be within. The circular portion of gastric wall which was outlined by the original incision is now excised and the posterior aspect of the anastomosis is completed with a row of fine silk sutures approximating the mucosal layers (Fig. 376). The transection of the esophagus is completed next and the diseased segment including a portion of the stomach and of the esophagus, is removed (Fig. 377). The completion of the anastomosis proceeds from this point by continuing the posterior layers around the circumference anteriorly. The sutures in the mucosal layer are inserted so that the knots are tied on the surface inside the lumen (Fig. 378). This is done to allow these sutures to separate and come away with greater ease. The middle and outer layers are placed in exactly the same manner as in the posterior portion of the anastomosis but in reverse order (Figs. 379 and 380). On its completion the anastomosis is exactly circular. If a tab of omentum is available on the greater curvature, this should be swung up and sutured around the anastomosis for additional protection.

ALTERNATIVE TECHNIQUE In certain instances when it is necessary to excise an unusually large portion of the stomach, leaving only a short distal fragment, an end-to-end esophagogastric anastomosis may be preferable. Although this method may be somewhat more difficult technically and subject to greater danger of inadequate healing, it is better in some instances than performing a total gastrectomy or leaving the turned-in distal segment and restoring continuity by esophagojejunal anastomosis. A large curved crushing clamp (Kocher) is placed across the stomach and near the greater curvature end a short straight clamp is put on to obviate the creation of a sharp angle at the end of the gastric remnant. The stomach is cut with a knife along these clamps. The open end of the distal portion is closed partially. The first layer consists of a continuous suture of fine chromicized catgut, starting at the lesser curvature and extending to the point where the unsutured remainder has a diameter which is approximately the equivalent of that of the esophagus. This corresponds to the portion outlined by the smaller clamp. Here the suture is tied. A second layer consisting of a continuous right angle stitch of the same material is inserted and an outer layer of Lembert sutures of silk is applied. All three layers are terminated before the greater curvature is reached, leaving an opening of adequate size for the anastomosis (Fig. 381, B). The anastomosis between the esophagus and stomach is then performed in the same manner as described above (Fig. 381, C, D, E, F).

Special pains must be taken to secure a satisfactory approximation of the layers at the point where the sutures used to narrow the open end of the remainder of the stomach meet the suture line of the anastomosis. It is the possibility of failure to secure prompt healing at this point which makes the end-to-end anastomosis more hazardous than the end-to-side which utilizes a circular opening in the stomach wall.

Tension on the anastomosis is prevented by fixing the stomach to the

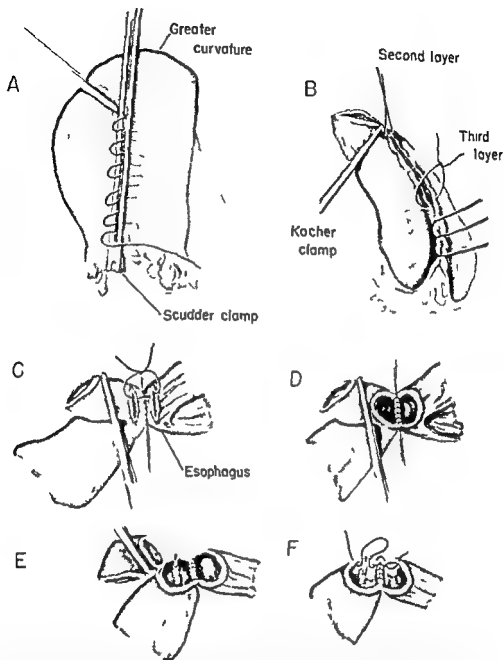


FIGURE 381 Alternative technique Anastomosis by end to end method *A* Fenestrated clamp in position at the level of transection of the stomach The start of the first suture line is shown, dotted lines indicate the portion of the suture which has been passed through to the other side of the stomach The suture is passed back and forth through the blades of the clamp A second clamp is then applied and the stomach is cut between A straight Kocher or Ochsner clamp is placed across the tip of the distal portion of stomach to outline the correct line of division for the anastomosis *B* Completion of the final layer for closure of part of the stomach *C*, Anastomosis first posterior layer completed second layer started after incision through the muscle layers *D* Posterior mucosal layer completed *E* First step in completion of anastomosis esophagus completely severed tip of gastric remnant almost entirely excised *F* First suture of the anterior extension of the mucosal layer showing the method of catching up both corners of the longitudinal gastric closure suture line

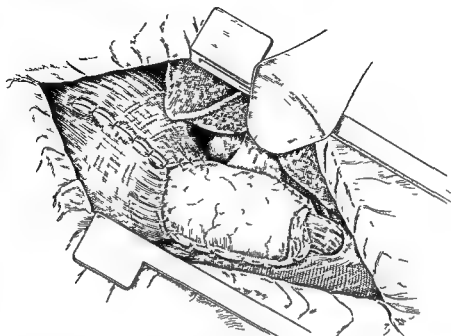


FIGURE 382 View of the mediastinum after completion of the operation showing closure of the diaphragm around the transplanted stomach the anastomosis with a tab of omentum from the greater curvature sutured over it for reinforcement and sutures between the stomach and the mediastinal pleura overlying the aorta

mediastinal pleura along the aorta with a row of silk sutures. The cut edges of the diaphragm are sutured to the stomach at an appropriate level below the anastomosis (Fig 382). In fitting the diaphragm to the stomach care should be exerted to avoid constriction. The opening through which the stomach is made to pass must be several times larger than the normal esophageal hiatus. The remaining edges of the diaphragmatic incision from the stomach wall to the costal insertion are then approximated using interrupted sutures of heavy silk.

Before the closure of the diaphragm is completed, a solution containing 1 gram of streptomycin and 100 000 units of penicillin in 30 cc of saline is injected, one half in the upper abdomen and one-half in the mediastinum and left pleural cavity. Closed catheter drainage is provided, using a Foley catheter brought out through the tenth intercostal space posteriorly.

Operation for Carcinoma of the Midthoracic Segment of the Esophagus. Partial Esophagectomy with High Intrathoracic Esophagogastric Anastomosis

This procedure offers better palliation and greater prospects of ultimate cure than the Torek operation which it now supplants in the treatment of carcinoma of the midthoracic region of the esophagus. It is a natural development resulting from modifications of the technique used for the removal of carcinoma located at or near the cardia. By preserving the right gastric and right gastroepiploic vessels and maintaining the continuity of the vascular communications along the greater and lesser curvatures and in the wall of the stomach itself an adequate blood supply may be preserved so that the entire stomach can be mobilized without danger of necrosis. After this has been accomplished and the

stomach has been cut across at the cardia, the fundus will reach as high as the apex of the chest and an anastomosis can be made at that level without danger of separation of the suture line. From the standpoint of the esophageal blood supply, it is important to remember that the segment of esophagus between the aortic arch and base of the neck is almost entirely dependent upon the esophageal branches of the inferior thyroid arteries. Whenever the high situation of the growth makes it necessary to mobilize the entire thoracic portion of the esophagus and to pull it from behind the aortic arch for a supra-aortic anastomosis, the level of division across the esophagus must therefore be at some point above the superior surface of the aortic arch. In order to avoid the development of necrosis at the cut end of the esophagus, this rule must be rigidly observed even though it appears that a greater useful length has been obtained as a result of such extensive mobilization.

After the thoracic and abdominal parts of the exploration have been carried out and the growth has been found to be suitable for resection, the freeing of the esophagus is completed from the diaphragm below to above the upper limits of the tumor. In the majority of instances this involves carrying the dissection up behind the aortic arch. Sometimes, however, a sufficient length of uninvolved esophagus exists between the growth and the aortic arch to make it possible to perform an anastomosis just below it. In a typical case the esophageal arteries from the descending aorta, the small esophageal branches of the bronchial and intercostal arteries, and the esophageal arteries arising from the aortic arch itself must all be divided.

The dissection between the hilus of the lung and the diseased area is often difficult. The freeing of an adherent tumor located behind the aortic arch is sometimes troublesome, but it can usually be accomplished by working from both above and below the arch. The dissection at this point may be facilitated by ligating and dividing the upper one or two left intercostal arteries as they arise from the aorta. This permits the aortic arch to be drawn forward sufficiently to complete what might otherwise be an almost impossible dissection.

In patients whose chest is long, access to the region above and behind the aortic arch may not be possible without enlargement of the incision by posterior division of one or more of the superjacent ribs. An alternative method of improving the exposure for this part of the operation consists in making a second opening through the bony thorax by resecting the fourth rib. This is done through the upper portion of the incision already made in the skin and muscle layers. Through this higher approach the aortic arch and the superior mediastinal portion of the esophagus are much more readily dealt with and the anastomosis more easily performed. Furthermore, the patient is left with a more stable chest wall and usually complains less of pain than when the usual type of incision with posterior upward enlargement through adjacent ribs is used.

Just above the superior surface of the aortic arch the thoracic duct is usually encountered as it crosses the esophagus to assume a more anterior location in the superior mediastinum whence it ascends into the neck. If the thoracic duct is adherent and a section of it must be excised with the growth, or if it is traumatized to the point where leakage of chyle might occur, it must be ligated. If this

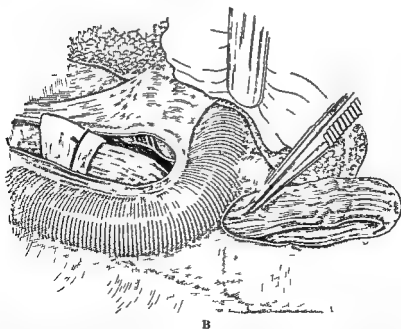
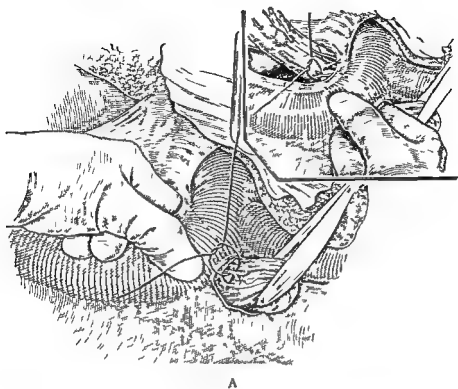


FIGURE 383 Stages in the dissection of the esophagus behind the aortic arch for the performance of a supra aortic anastomosis *A* Ligation of retro-aortic vessels blunt dissection with the finger *B* Withdrawal of the esophagus (J Perrotin)

is not done, a fatal outcome is almost certain to result from *inanition due to the loss of chyle*

During the dissection of an adherent growth, the right pleural sac is often opened unavoidably. A portion of the mediastinal pleura and occasionally of the right lung itself may have to be excised. If the lung is cut across, the defect is sutured with a continuous suture of fine catgut. No attempt need be made to close the opening in the right pleural reflection because at the completion of the operation both lungs will be expanded by the positive pressure within the anesthesia system, and this expansion is maintained by the intrathoracic negative pressure after the wound has been made air-tight by closure.

After the dissection of the esophagus has been completed, the incision in the diaphragm is enlarged through the hiatus and the entire stomach is mobilized almost as extensively as where a total gastrectomy is to be performed, with the vital exception that the right gastric and right gastro epiploic vessels are preserved, together with the anastomotic arches along the curvatures. The continuity of the anastomotic arches along the lesser curvature depends upon the fact that the left gastric artery, with which the branches of the right gastric artery communicate to make a continuous channel, must be severed as close as possible to its origin from the celiac axis. Division of the stomach is carried out as close to the cardia as is consistent with the removal of the lymph nodes in that region. The distal cut edge of the stomach is inverted by the technique previously described. An alternative method, which can be used when the growth is so high that it is important to avoid the sacrifice of any gastric vessels, is to tie the esophagus 2 cm. above the cardia with a ligature of heavy silk and to invert the stump after transection by means of a purse string suture in the stomach at the level of the cardia. Further reinforcement is provided by means of a layer of Lembert sutures. This method is unsatisfactory if lymph node metastases are present in the region of the cardia.

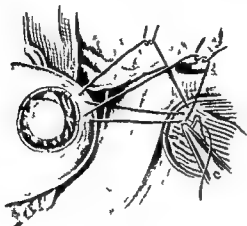
In handling the stomach, great care must be exerted to avoid trauma to any of the intercommunicating anastomotic branches of the vessels in its walls, especially in the fundus where no extrinsic vessels exist.

The proximal cut end of the esophagus is covered by a rubber glove or a square of rubber tissue tied over it. The esophagus is then pulled up from behind the aortic arch and the stomach is brought up into the apex of the chest for the performance of an anastomosis (Fig. 383, B). The circular incision in the fundus of the stomach is prepared and the esophagogastric anastomosis is performed in exactly the same manner as when the carcinoma is at the cardia. Because of the high level at which the anastomosis must be made, unusually long instruments are required.

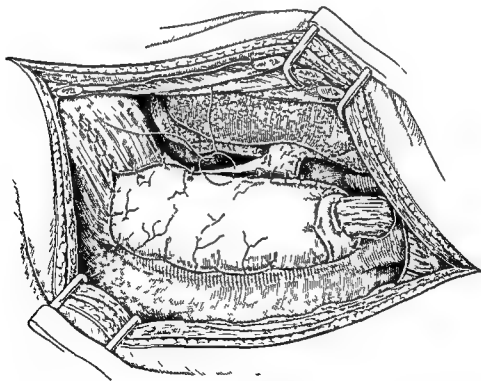
The stomach can be brought up to meet the esophagus for a high anastomosis either to the left and in front of the aortic arch (Fig. 384) or to the right and behind the arch. If the latter technique is used, the stomach is grasped by the surgeon's left hand and introduced behind the aortic arch from below. The portion of the gastric wall which has been outlined by circular incision into the muscularis in preparation for the anastomosis is grasped with a Collin forceps or other suitable nontraumatizing instrument and drawn up behind the arch into the superior mediastinum (Fig. 385). By exerting gentle traction with

the grasping forceps a sufficient excess of stomach wall is drawn forward to make it possible to perform the anastomosis in the manner described above

The advantage of bringing the stomach up in this manner is that it lies in the mediastinum rather than in the pleural cavity and therefore tends to interfere less with complete expansion of the lung. At the completion of the anastomosis,



A



B

FIGURE 384 Steps in the performance of a supra aortic anastomosis stomach to the left of the arch in the left pleural cavity. *A* Start of anastomosis. *B* Anastomosis completed diaphragm closed stomach supported with sutures to the pleura overlying the aorta (*A* Sweet *B* J Perrotin)

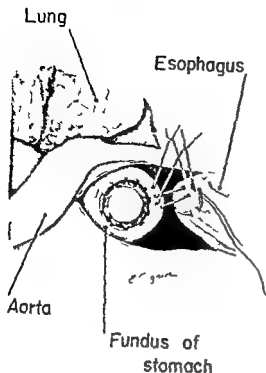


FIGURE 385 Start of the supra aortic anastomosis with stomach drawn up to the right of the aortic arch through the mediastinum

in order to hold the stomach in position, a suture of fine silk is placed on either side between the fundus and the adjacent edge of the incision in the pleural reflection over the superior mediastinum. The remainder of the stomach is fixed within the chest by means of a long row of sutures to the pleura overlying the thoracic portion of the descending aorta. The closure of the diaphragm about the stomach is completed as described previously (Fig. 382). The pylorus usually lies only a few centimeters below the diaphragm at the completion of the operation. The antibiotic solution is injected both above and below the diaphragm as already described.

Operation for Carcinoma of the Superior Mediastinal Segment of the Esophagus **Subtotal Esophagectomy with Intracervical Esophagogastric Anastomosis**

The resection of a carcinoma in the superior mediastinal segment of the esophagus presents the most difficult technical problem of all possible locations. The high situation of the growth makes it necessary to transect the esophagus through its short cervical segment, thus removing almost the entire length of the esophagus. This entails the making of a separate cervical incision. Furthermore, it is necessary to provide a means of passing the completely mobilized stomach from the thorax into the neck where the anastomosis must be performed.

This step may be completed in one of two ways. If the stomach is not too large, it is possible to bring it up behind the aortic arch and into the superior mediastinum through the thoracic inlet in the bed from which the esophagus is removed. The employment of this method, however, involves the possibility of irreparable damage to the blood supply of the fundus of the stomach because of the amount of manipulation required or the possibility of ischemia of the

fundus because of the constriction produced by forcing it through too small an aperture. This technique is more likely also to be complicated by trauma to the recurrent laryngeal nerves, particularly the right, with attendant respiratory difficulties. In the average case, therefore, it is probably safer to create an artificial channel from the left thoracic cavity into the neck by excising the inner portions of the clavicle and the first rib. It should be recognized that the exact details of this part of the procedure are not yet well established and may be modified subsequently as more experience is acquired.

The operation using the latter method consists of two steps performed in one stage as follows:

FIRST STEP Dissection of the Esophagus and Mobilization of the Stomach

A standard thoracotomy incision is made on the left side, resecting the eighth rib. If sufficient exposure is not available to reach the superior mediastinum, the seventh, sixth, and sometimes the fifth ribs may be divided posteriorly or a higher rib (usually the fourth) may be resected, producing a second opening through which the superior mediastinum can be more easily exposed. The dissection is begun in the region of the tumor after incising the mediastinal pleura above the aortic arch and posterior to the left subclavian artery. If the tumor-bearing portion of the esophagus can be freed sufficiently to make it possible to perform a resection, the operation is continued by completing the dissection of the entire length of the thoracic portion of the esophagus. The freeing of the superior mediastinal segment presents no particular problem except that the thoracic duct must be identified as it crosses the esophagus just above the aortic arch. If the duct is invaded by tumor or is too adherent to be dissected free or if it has been injured during the dissection, it must be ligated to prevent the development of a chylous hydrothorax after the operation. Below the aortic arch the esophageal arteries must be tied and cut. In freeing the lower portion of the esophagus, the periesophageal lymph nodes must be included in the dissection insofar as possible. After the entire thoracic portion of the esophagus has been dissected free, the phrenic nerve is crushed and the diaphragm is incised from a point close to its costal insertion through the margin of the esophageal hiatus. This permits the complete mobilization of the stomach, which is carried out exactly as in the cases of esophagectomy for carcinoma of the midthoracic segment of the esophagus requiring a supra-aortic anastomosis.

After the mobilization of the stomach has been completed, the esophagus is divided at or close to the cardia and the end on the gastric side is either inverted with a purse-string suture reinforced with a layer of Lembert sutures of silk or sutured as described above. Care must be exerted to avoid injury to any of the vessels of the anastomotic communications in the region of the cardia. A piece of rubber tissue or a rubber glove is tied over the proximal end and the esophagus is then pulled up and out from behind the aortic arch into the left pleural cavity so as to make its subsequent withdrawal into the neck somewhat easier.

The stomach is now drawn up as far as possible into the left side of the thorax behind the hilus of the left lung and lateral to the aortic arch. Fixation in this location is maintained by means of a series of interrupted silk sutures

between the gastric wall and the mediastinal pleural surface which overlies the descending aorta. The fundus of the stomach is allowed to be free in the superior portion of the pleural cavity until the cervical portion of the operation is performed. The fixation sutures already applied prevent its falling down out of reach after the chest has been closed. The diaphragm is sutured to the stomach just above the pylorus and the remainder of the diaphragmatic incision is closed after injecting the antibiotic solution, as described in the section dealing with the technique of partial esophagectomy at lower levels.

A Foley catheter is led out through a small incision in the tenth intercostal space posteriorly. To complete the first step of the operation the lung is expanded by the anesthetist and the thoracotomy incision is closed using interrupted silk sutures in all layers.

SECOND STEP *Performance of the Intracervical Esophagogastric Anastomosis*
After closure of the thoracotomy incision has been completed in the usual manner, the patient is turned on his back and an incision is made along the anterior margin of the left sternocleidomastoid muscle to the suprasternal notch and then downward over the upper portion of the sternum to the level of the second costal cartilage. The pectoralis major muscle is incised close to its sternal attachment and reflected laterally, at the same time severing its medial clavicular insertion. The sternal and medial clavicular insertions of the sternocleidomastoid muscle are severed and retracted laterally. The dissection is deepened in the space between the carotid sheath and the trachea to expose the esophagus, which is grasped with a forceps and pulled up from the mediastinum and out in front of the carotid sheath.

The medial half of the clavicle and a corresponding segment of the left first rib and costal cartilage are excised extraperiosteally. The clavicle should be cut with a Gigli saw. This produces a large opening from the base of the neck into the apex of the left pleural cavity through which the fundus of the stomach can be drawn easily and without danger of compression. The pleura is opened and the fundus is brought forward medial to the apex of the lung and pulled up into the lower portion of the neck through this channel (Fig. 386). A short linear incision is made in the posterior wall of the fundus close to its apex and an anastomosis consisting of three layers of interrupted fine silk (5-0) sutures is made. Careful approximations of mucosa to mucosa and of muscle edge to muscle edge constitute the inner and middle layers. The outer layer is of mattress sutures. Several sutures are used to fasten the fundus to the surrounding tissues of the neck to prevent tension on the anastomosis which, after its completion, lies in front of the carotid and internal jugular vessels. The wound is closed by suturing the lower end of the sternocleidomastoid muscle and the medial cut edge of the pectoralis major muscle to the sternum and placing a layer of fine silk sutures in the subcutaneous fat and another in the skin. Drainage is not employed.

CARE OF THE PATIENT AFTER ESOPHAGECTOMY AND ESOPHAGOGASTRIC ANASTOMOSIS

The administration of streptomycin and penicillin is continued by intramuscular injection during the first five days after operation or until any suspicion

of infection in the lungs or within the abdominal or pleural cavities or in the mediastinum has subsided. A satisfactory dosage is 0.25 gram of streptomycin and 100,000 units of penicillin every six hours. Sulfonamides are no longer used.

Administration of oxygen by the intranasal method is employed during the first twenty-four hours. After that time it may be omitted in the average case. In patients who have had a supra aortic or intracervical anastomosis, the large incision required and the extensive dissection within the mediastinum, as well as the presence of almost the entire stomach within the left thoracic cavity or mediastinum make the readjustment of the respiratory and circulatory functions more difficult. As a result the inhalation of oxygen may have to be continued at least intermittently for three to five days.

In the patients with an anastomosis low in the thorax, the *Levin tube* is left at the completion of the operation with its tip just proximal to the anastomosis in order to avoid the possible danger of necrosis at the suture line which might

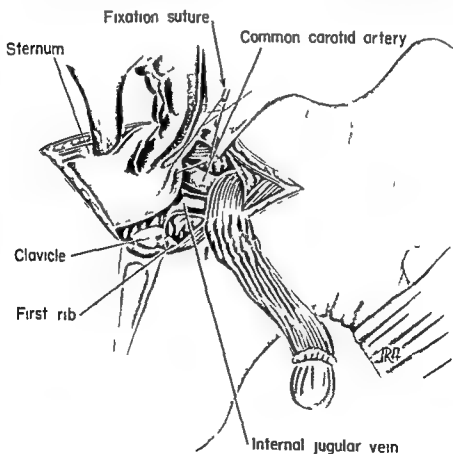


FIGURE 386 Intracervical anastomosis for carcinoma in the superior mediastinal segment. The esophagus has been pulled up from the mediastinum and brought out in front of the carotid sheath. The fundus of the stomach has been drawn up through the left pleural cavity and into the neck by way of an opening created by the excision of the inner third of the left clavicle and a corresponding portion of the left first rib.

result from the pressure of the tube if it were passed through into the stomach. The amount of drainage obtained by continuous aspiration of the Levin tube is usually small and the tube can therefore be removed in the average case after twenty-four hours.

In patients who have a supra-aortic or intracervical anastomosis, because of the danger of sudden regurgitation of large amounts of gastric contents and the disturbance of the respiratory and circulatory functions which may result from the pressure of an overdistended intrathoracic stomach, it is preferable to overlook the possible disadvantages of the presence of a tube and to leave the Levin tube with its tip actually within the stomach at the completion of the operation. Continuous aspiration is then maintained for a period of three to five days before the tube is removed. Thereafter it may still be necessary to reinsert a tube temporarily if gastric dilatation should occur, but this is an infrequent occurrence.

The intrathoracic drainage catheter is removed after forty-eight hours. Subsequent removal of fluid by thoracentesis is rarely necessary when the antibiotics are used prophylactically. The reaccumulation of a large amount of fluid after the first effusion is over usually heralds the onset of empyema, a rare occurrence at the present time.

In patients 50 years of age or older the routine administration of quinidine and avoidance of the overadministration of fluid intravenously are applied as in any other patient of like age who is subjected to a major thoracic operation.

During the first few days the patient's requirements of food and fluid are maintained almost entirely by intravenous injection. By this means glucose, amino acids, vitamins, and whole blood, if indicated, are administered, taking care always to avoid embarrassment of the circulatory system.

The oral administration of fluids is begun after twenty-four hours, with 30 cc of water each hour. The second day after operation 60 cc of clear fluids, not including fruit juices, may be given hourly. It is unwise to increase this amount, however, on the third day or even, with some patients, on the fourth. By the fourth or fifth postoperative day liquids made with milk may be added and slightly larger amounts administered each hour. From that day on the amount and character of the feedings can be adjusted to suit the ability of the patient to take them. Gradually the amount per feeding is increased and the intervals between them lengthened. The patient should not be urged to eat, however, until he feels the inclination to do so or at least until after the expiration of ten to twelve days, when the danger of damage to the anastomosis from overloading the stomach is not great. The majority of patients, however, are able to eat a soft solid diet divided into six small meals per day by the twelfth postoperative day.

Patients who have had an esophagectomy are well enough usually to get up from bed on the second postoperative day. Many of them are allowed out of bed on the first day after operation. A few patients, however, must be kept in bed four or five days before the readjustment of the respiratory and particularly the circulatory function is sufficiently well established to allow greater activity. This rule applies almost exclusively to patients with a growth which necessitates the performance of a high supra-aortic arch anastomosis or one within the neck.

THE MANAGEMENT OF THE LATE RECOVERY PERIOD

The majority of patients on whom an esophagectomy has been performed (excluding those whose growth lies in the cervical segment) are able to return home within two to three weeks after operation, depending upon the distance to be traveled and the possibilities of care at home. Thereafter there is a period of readjustment which may present difficulties and lead to anxieties from which the surgeon should seek to protect the patient by explanation and advice.

In common with patients who have been subjected to a total gastrectomy, patients who have had operations of the sort described frequently complain that they do not regain a normal appetite. This may correct itself after weeks or months have elapsed but in many instances the return of appetite is incomplete at best. This occurrence is associated with and possibly explained in part by the fact that many patients experience a functional delay in the emptying of the stomach. The interruption of the vagus nerves is a contributing factor because of the resulting diminution in the amplitude of the gastric peristaltic activity and because of the hypertonicity of the pyloric sphincter. The result is that the stomach remains partially filled much of the time. It is a common observation among these patients that they are able to eat a large breakfast but that they have little appetite for their noonday meal and are able to accommodate hardly any of their supper. This functional difficulty is most pronounced in patients with carcinoma of the cardia which requires excision of a large segment of the stomach. This of necessity leaves only a small distal portion which accommodates a limited volume of food. It has been observed, however, that pronounced examples of the inability of the stomach to empty after the performance of a partial gastrectomy and esophagectomy have become much less frequent than formerly and that the difficulty arises least often in patients who have had a high esophagectomy. A possible explanation for this is that the division of the gastrosplenic and gastrohepatic ligaments is now carried all the way to the level of the pylorus in every instance thus probably interrupting many of the sympathetic fibers which would otherwise be overactive because of the absence of the vagus inhibition.

In evaluating the postoperative digestive function of these patients it should be kept in mind also that, with the exception of lesions high in the esophagus where the whole stomach is preserved, it is necessary to resect portions of the fundus of the stomach. This sometimes causes a large reduction in gastric volume which further limits the ability of the patient to take food.

The inability of patients to handle large quantities of food during the first few months of their convalescence is sometimes so great that there is a progressive loss of weight. Such patients should be advised to take nothing but the most nourishing types of food and to avoid wasting valuable space on materials either liquid or solid which have a low caloric content. As time goes on, however, in the majority of instances the patient's capacity for food increases as the gastric remnant enlarges and the emptying time of the stomach approximates a normal rate. In spite of this it is unusual for the patient to regain his customary weight. More often he will gain only a few pounds. Many times he will be able merely to hold his weight at a reduced level without further loss.

Although it is frequently necessary to ligate the thoracic duct particularly

in the cases in which the position of the growth requires a supra-aortic arch anastomosis, no disturbance of nutrition which can be attributed to the procedure need be anticipated. The nutritional status of such patients does not vary in any respect from that of those whose thoracic duct remains undisturbed.

A troublesome occurrence frequently observed is the tendency to regurgitation from the stomach if a recumbent posture is assumed soon after eating. Patients should be advised not to lie down during the first two hours after meals. In some it is never possible for them to lie on their right side without experiencing regurgitation.

Occasionally a patient may develop diarrhea which lasts sometimes a few days, sometimes several weeks before it subsides. This is probably caused by the disturbance of function resulting from bilateral vagus section. However, this phenomenon is observed in these patients much less frequently than among those who have had a vagotomy performed in the treatment of duodenal ulcer. Relief is obtained by means of the usual symptomatic treatment.

Recurrences of dysphagia are exceedingly unusual after esophagectomy with esophagogastric anastomosis. Cicatricial stenosis of a properly performed anastomosis is a rare occurrence. Treatment by bougienage is usually sufficient to overcome the difficulty if it should occur. Recurrence of carcinoma at the anastomosis is observed occasionally, but the majority of the patients who succumb to the disease die from the effects of distant metastases or local recurrence within the mediastinum, and retain their ability to swallow normally as long as they live.

Although a large portion of the left thoracic cavity may be occupied by the transplanted stomach, patients on whom an esophagectomy has been performed almost never experience any sensations which might make them aware of the presence of the stomach within the thorax. Gastric peristaltic sounds are occasionally heard, but they are more often noticed by other people than by the patient himself. Furthermore, after the immediate postoperative period of readjustment has gone by, there is rarely any striking dyspnea or evidence of circulatory disturbances.

Operation for Carcinoma of the Cervical Segment Wookey Procedure

Technique

FIRST STAGE: Endotracheal inhalation anesthesia is used. The patient is placed on his back in the position used for thyroidectomy, with moderate hyperextension of the neck. A long rectangular incision is made across the neck, outlining a flap of skin with its base attached on the right and its free end extending to the anterior edge of the left sternocleidomastoid muscle. The upper line of the incision starts just below the angle of the mandible and the lower line just above the clavicle. The two lines are made to converge slightly so that the base of the flap will be approximately 1 inch wider than its free end, which should be about 3½ to 4 inches in the average case. This three-sided incision is deepened through the subcutaneous fat and the platysma muscle. The flap is then dis-

sected back in the layer beneath this muscle to the region of the posterior edge of the right sternocleidomastoid muscle. The flap thus consists of skin, subcutaneous fat, and platysma in one thickness (Fig. 387). It is laid between two gauze pads moistened in saline solution. A plane of dissection is developed along the anterior border of the sternocleidomastoid muscle and the prevertebral fascial space is entered, exposing the cervical segment of the esophagus where the growth lies. To obtain an adequate exposure longitudinally, it is necessary to transect the omohyoid muscle. If the growth is small and does not invade the layers and if it is not surrounded by lymph nodes involved in carcinomatous metastases, the way is prepared for its removal. The dissection is carried up as far as the hypopharynx and down to the superior mediastinum. The superior and inferior thyroid arteries and one or two pharyngeal and upper esophageal arteries are severed. The corresponding veins are divided. The sternal attach-

First Stage

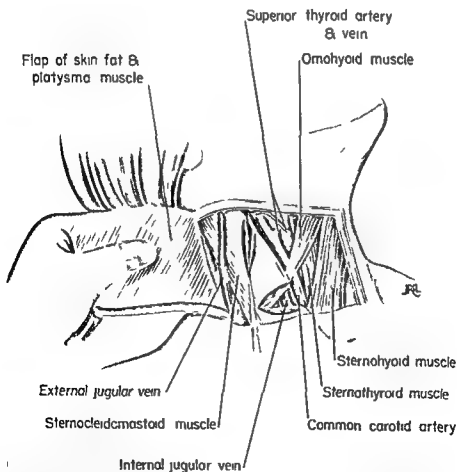


FIGURE 387 Cervical esophagectomy. Wookey technique. First stage. Some of the important structures exposed by the reflection of a rectangular flap of skin fat and platysma muscle are shown. Note excision of a triangular piece of the sternocleidomastoid muscle.

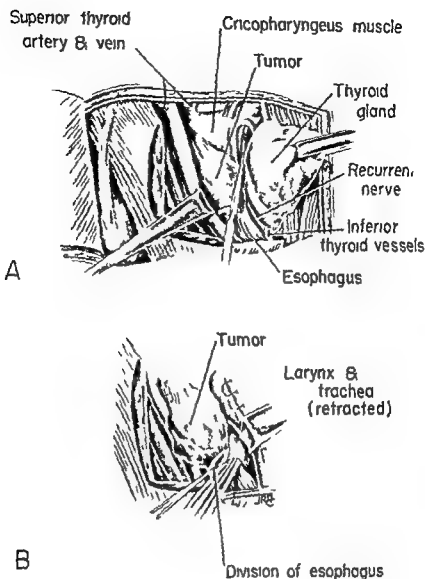


FIGURE 388 : Cervical esophagectomy First stage (continued) A Exposure of the tumor and excision of right lobe of the thyroid gland B Division of the esophagus below the growth

ment of the sternocleidomastoid muscle is cut close to its insertion into the bone and a long triangular-shaped section of the muscle belly is excised (Fig 387) This is to facilitate the proper fitting of the flap against the prevertebral fascia in that area The right sternohyoid and sternothyroid muscles are excised and the right lobe of the thyroid gland is removed, cutting across the isthmus just to the left of the trachea (Fig 388) During this part of the procedure the right recurrent laryngeal nerve is exposed in the lower part of the field of operation and, unless it is invaded by tumor, should be preserved

The esophagus is separated from the trachea and a piece of tape or narrow Penrose drain is passed around it to be used for retraction The dissection between the esophagus and the prevertebral fascia is continued up to the pharyngeal region The esophagus is cut across as far as possible below the growth

be difficult to suture the lower edge of the flap to the lower limit of practicability is approximately at the of the clavicle. The transection above the tumor is ophageal junction. Rarely it is possible to preserve ip of esophagus, but only in the case of a low-lying vessels which approach the esophagus from the left. A strand of fine silk is passed with a needle through of the lower segment to provide traction.

a muscle flap is now turned over into the defect and p rows of fine chromic catgut (5-0) sutures between the platysma and the prevertebral fascia. The right lateral row is inserted first and tied before the left lateral row is put in (Fig 389 A). This maneuver fixes

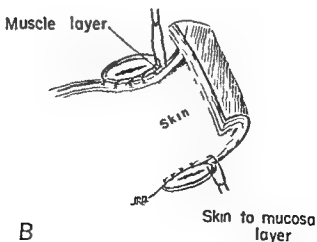
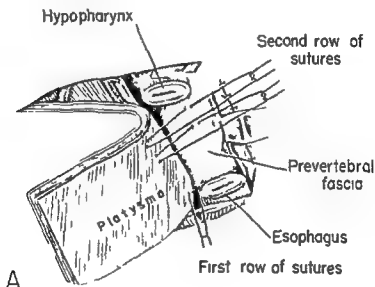


FIGURE 389 Cervical esophagectomy. First stage (continued). A Suture of the flap to the prevertebral fascia. B Suture of the edges of the flap in two layers to the posterior margins of the pharynx above and the esophagus below.

the posterior surface of the flap. The next step is to attach the upper edge of the flap to the pharynx and the lower edge to the esophagus. This is done with two layers of sutures. The outer or peripheral layer approximates the platysma muscle and the muscularis of the pharynx and esophagus. The inner layer joins the skin and the mucous membrane of the two organs. Interrupted sutures of fine chromic catgut are used. The posterior muscle layer is inserted first both above and below as far as the left corner on each edge. When this point is reached, the approximation of the skin and mucosa is made. The same suture material is used, but small, fine, cutting point needles are needed to pierce the skin (Fig. 389, B).

After the posterior inner layers are completed, the approximation of the anterior edges is begun by continuing the mucosa-to-skin layer both above and below. This is continued around to the right as far as the starting point on the circumference of the esophagus and pharynx. The muscle layer is inserted next (Fig. 390, A). The remaining free end of the flap is then turned to the left over the larynx and the trachea. Its edges are sutured in layers to the upper and lower edges of the incision as far as the tip of the flap. The defect on the left side of the neck which results from the shortening of the flap produced by folding it into the depths of the neck is covered by a primary Thiersch graft obtained from the skin of the left thigh by an assistant. The superior and inferior edges to the right of the infolding of the flap are sutured, also in two layers. Silk is used in the skin (Fig. 390, B).

As a result of the surgical trauma in the region of the pharynx and larynx a considerable degree of edema and swelling of the tissues develops, reaching a maximum sometime between twelve and forty eight hours after the completion of the operation. The swelling is sometimes so great that serious obstruction of the airway through the larynx results and an emergency tracheostomy may become necessary. It is preferable, therefore, to perform a tracheostomy as a part of the primary procedure in all cases. This is done through a short incision in the skin just above the suprasternal notch (Fig. 390, B). The tube is held in place by means of tapes tied around the neck in the usual manner.

A sterilized Levin tube is inserted into the stomach through the lateral groove produced by the infolded flap.

Aftercare The aftercare following the first stage of the operation presents no special problems. The antibiotics should be continued for five to seven days postoperatively. Feeding through the Levin tube is begun as soon as the patient has recovered sufficiently from the effects of the anesthetic, usually on the subsequent day. The patient's nutrition is provided by a liquid gastrostomy formula during the interval between the stages.

The tracheostomy tube is removed after the danger of obstruction of the airway from laryngeal edema has subsided. This is usually on the third to the fifth day.

SECOND STAGE The second stage must not be attempted until the wound is completely healed and the subcutaneous tissues are soft and pliable. This also allows enough time to elapse for the implanted skin to acquire a blood supply from the bed in which it lies. The usual interval of time is approximately two months.

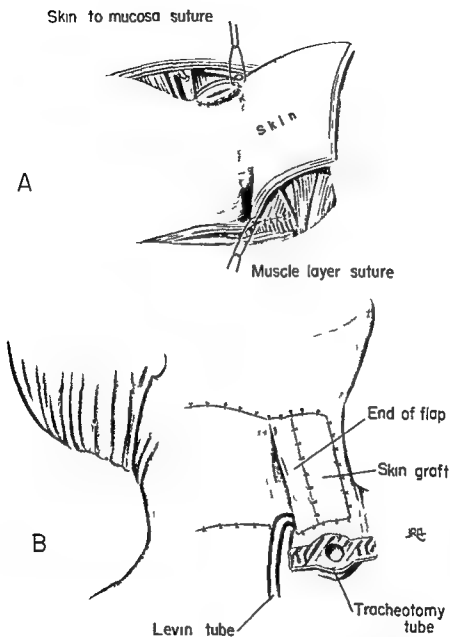


FIGURE 390 · Cervical esophagectomy First stage completed. *A* Completion of the flap to the pharynx and esophagus *B* Completion of skin closure using skin graft Levin tube for feeding Tracheostomy tube to avoid suffocation from laryngeal edema to be removed after five to seven days

Second Stage

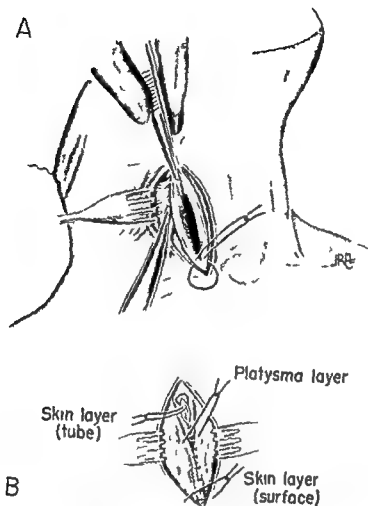


FIGURE 391 Cervical esophagectomy second stage *A*, Incision around the lateral cleft *B* Two layers of sutures close the skin tube. Innermost layer of fine chromic catgut (5-0) in the edges of the skin tube, next layer approximation of platysma (catgut), outer layer of skin closed with silk

The patient is anesthetized in the usual manner. The Levin tube is removed and replaced by a second tube which is inserted through the nose and threaded down the pharynx through the skin groove, into the esophagus and stomach. An elliptical incision is made around the periphery of the lateral groove, allowing a sufficient margin for closure (Fig. 391, *A*). The inner edges are approximated with a layer of fine catgut sutures inserted so that the knots, when tied, are inside the lumen. A second layer consisting of connective tissue and muscle is brought together over this. The outer edges of the wound are approximated in layers in the same manner (Fig. 391, *B*).

After recovery from anesthesia the patient is fed for a period of approximately seven to ten days by means of a Levin tube inserted through the nose. The tube is then removed and oral feeding is begun. The patient does not as a rule experience any difficulty with deglutition (Fig. 392).



FIGURE 392 Roentgen film showing barium in the upper esophagus before and after operation. *A* Lateral view showing large tumor partially occluding the lumen and bulging into the posterior wall of the trachea. *B* Same patient after operation. lateral view showing excellent functional result of skin tube substitution.

Complications and Operative Mortality after Esophagectomy

Complications after Cervical Esophagectomy

Complications following the Wookey operation in the removal of a carcinoma of the cervical segment are rarely serious. Postoperative pneumonia is seen occasionally, but this and other respiratory complications are kept at a minimum by the routine insertion of a tracheostomy tube at the completion of the first stage. This maneuver avoids obstruction of the airway due to laryngeal edema and facilitates the removal of secretions from the trachea and bronchi during the first few days. Antibiotics are of course employed.

Local inflammation and swelling of the tissues of the neck resulting from the unavoidable contamination of the operative field with mouth organisms is an almost invariable occurrence after the first stage. Spreading sepsis, thanks to antibiotic medication, is not observed.

Occasionally there may be *loss of the tip of the skin flap* from inadequacy of its blood supply or a part or all of the skin graft on the left side of the neck may not take. Further use of skin grafting when the condition of the tissues is favorable makes up for these deficiencies.

Leakage after closure of the opening which comprises the second stage of the procedure has not been observed. It is important in this regard not to yield to the temptation to perform the closure too soon after the first stage, before the inflammatory swelling has gone from the tissues and before the skin flap has acquired a blood supply from its new bed.

The mortality after the Wookey operation is not over 3 per cent. The one postoperative death in the series to be reported here was from cerebral hemorrhage.

Complications Occurring after Esophagectomy for Carcinoma of the Thoracic Portion of the Esophagus

These may be considered under the following headings: (1) those having to do with technical faults of the operation, (2) those of the lungs and pleural cavity, (3) those of the heart, and (4) miscellaneous complications not having a specific relation to the type of procedure.

COMPLICATIONS DUE TO TECHNICAL ERRORS: Under this heading are chiefly *leakage with fistula formation and stricture of the anastomosis*. The cause of leakage is invariably failure of the blood supply either of the residual end of the esophagus which has been employed in the making of the anastomosis or of the stomach. The tenuous nature of the segmental blood supply of the esophagus makes it essential always to divide the organ close to the last available intact artery. The principal difficulty arises from a failure to divide the esophagus well above the level of the aortic arch whenever it is necessary to perform a supra-aortic anastomosis. It often happens that an appreciable length of uninvolved esophagus at the aortic arch level is available for use, but the surgeon should never yield to the temptation to leave this additional length, because of the inadequacy of its blood supply. As was pointed out above, severing of all its vascular connections in the region of the arch and the bronchi leaves this portion of the esophagus devascularized because the branches of the inferior thyroid artery do not reach down to this level. Under these circumstances the esophagus must be divided relatively high in the superior mediastinum.

In the majority of instances, however, the failure is on the gastric side. This stems from the fact that in some way the surgeon has neglected to preserve adequate blood supply for every portion of the mobilized stomach. In every case both the right gastric and the right gastroepiploic arteries must be preserved. In every case, also, the integrity of the vascular arches of the gastroepiploic system along the greater curvature must be preserved. With a low esophageal or cardiac tumor, it is necessary to remove the proximal portion of the lesser curvature along with all of the lymph nodes in the left gastric area. This means that the ascending branches of the right gastric artery must be severed where they intercommunicate with the descending branches of the left gastric artery. Division of the stomach close to this level and removal of the entire cardia and fundus, however, automatically avoids leaving any gastric wall which may become necrotic (Fig. 393, 3).

On the other hand, with a carcinoma in the midesophagus or higher where, in order to gain adequate length, it is necessary to preserve the entire fundus of the stomach, it is absolutely essential to maintain the integrity of the intercommunicating arches along the lesser curvature as well as those along the greater curvature. This must be done even at the expense of leaving some of the lesser curvature lymph nodes which cannot be removed under these circumstances.

To accomplish this the left gastric artery must be divided close to its origin

and the ascending branches reaching up to the cardia must be preserved (Fig 393, 2) If this precaution is not observed, leakage from the anastomosis or from a focal area of necrosis in the wall of the fundus distant from the anastomosis is likely to result

Another fault which may end in leakage from an area of necrosis of the gastric wall is failure of the blood supply of the strip of stomach between the closed-in end and the site of the anastomosis This occurs when the latter has been placed too close to the inverted end of the stomach

The second complication which results from technical errors is the occurrence of a *stricture of the anastomosis* within the first few weeks or months after the operation The details of technique which are necessary to avoid this occurrence are those which reduce to a minimum the degree of trauma to the divided edges which are to be employed in the making of the anastomosis The

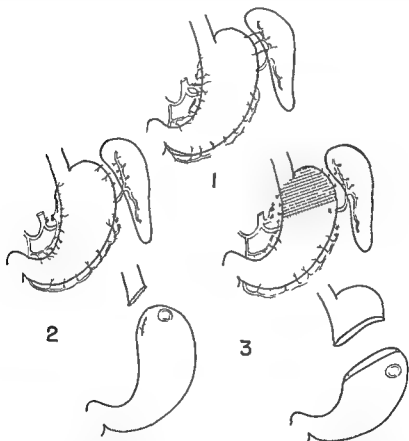


FIGURE 393 Diagram illustrating the principles involved in the avoidance of inadequacy of blood supply of the gastric fundus in esophagectomy using the stomach for replacement 1 Blood vessels of the stomach before mobilization for esophagogastric anastomosis 2 Division of vessels as it should be done if the entire stomach must be utilized (high mediastinal or intracervical anastomosis) Note preservation of both descending and ascending (cardial) branches of the left gastric artery leaving an intact arcade of intercommunicating vessels along the lesser curvature anastomosis using the fundus as shown perfectly safe 3 Cases requiring division of the ascending branches of the left gastric artery in order to remove the left gastric lymph node group as shown demand removal of the cardia and fundus of the stomach in order to avoid necrosis on the gastric side of the suture line Anastomosis must be placed where shown.

tissues must be cut with a sharp instrument (knife or scissors), not with a cautery, no clamps (crushing or otherwise) should be applied to the edges which are to be sutured, and the layers of the anastomosis must be made with interrupted sutures, not with a continuous strand which when pulled tight causes pressure necrosis of the edges in addition to the tendency to narrow the stoma by shirring.

PULMONARY COMPLICATIONS Pulmonary complications are among the most serious and the most numerous of all the postoperative complications after esophagectomy for any lesion, but especially in the elderly and often debilitated patients who are afflicted with esophageal carcinoma.

Atelectasis of the lungs is the most frequent occurrence. This may appear on one or both sides at once. It is often scattered over many small areas, but it may be massive in individual segments or lobes. The cause is invariably plugging of the bronchi with retained mucoid secretion which the patient is unable to raise and expectorate. Because of the interference with the diaphragm and the presence of the thoracotomy incision, both of which tend to inhibit full inspiration and expiration on the side of the operation, the left lung is likely to be more often involved than the right.

Adequate medication to control incisional pain must be administered but not to the point where the respiratory function is inhibited. If the pain is severe, the relief obtained from injecting the intercostal nerves in the region of the incision with procaine hydrochloride will often tide over a critical period and permit the patient to breathe and to cough more effectively. Steam inhalations and the exhibition of expectorants to loosen the secretions may be helpful.

When the piling up of mucus in the trachea and bronchi reaches the point where breathing is seriously interfered with, more active measures must be taken to prevent hypoxia which if prolonged may lead to serious consequences. To empty the respiratory passages quickly a *bronchoscopic aspiration* can be performed with the patient in his bed in the hospital room. This is effective for short periods of time, but when the secretions continue to accumulate, resort to repeated bronchoscopies becomes impracticable. When this situation arises, it is preferable to perform a *tracheostomy* through which the secretions may be aspirated at frequent intervals by the nurse. This is often a life-saving procedure and should not be delayed whenever the need is apparent.

In some instances aspiration through a catheter inserted into the trachea by way of the larynx is successful, but the method requires unusual skill on the part of the attendants and cannot always be used.

Difficulties with accumulating secretions are greatest in patients who have had an extensive esophagectomy with a supra aortic anastomosis and particularly in those whose anastomosis has to be made in the neck. In the latter group it is expedient to insert a tracheostomy tube routinely at the completion of the operation.

Elderly and weak debilitated patients are those most subject to this complication.

Frank pneumothorax is much less frequent than atelectasis. It is usually bronchial and multifocal in distribution, often bilateral. *Lobar pneumonia* is not frequent. Treatment with appropriate antibiotics is indicated.

Empyema since the routine use of antibiotics was adopted has become a most unusual complication. Its development is heralded by the reaccumulation of fluid in the chest once the first effusion has been removed. It often apparently follows unrelieved atelectasis, although it may occur independently. Differentiation between the two conditions is not always possible unless there is clinical and particularly roentgen evidence of rapid clearing of an acute pulmonary process. If uncontrolled by antibiotics appropriate measures must be instituted to provide proper drainage according to the accepted principles of treatment.

Pulmonary edema is a fairly infrequent occurrence usually as a manifestation of cardiac decompensation. It demands vigorous treatment according to the requirements of the individual patient.

CARDIAC COMPLICATIONS Cardiac complications are next in importance and frequency to those involving the lungs. Because of the relatively advanced age of the majority of the patients a comparably high incidence of *myocardial infarction* and *disturbances of rhythm* must be expected. This should prompt the surgeon to seek the advice of a cardiologist in the management of these patients both as to the prophylaxis and the treatment of possible abnormalities. Auricular fibrillation is the most frequent arrhythmia encountered and usually the most amenable to treatment using digitalis.

A more serious disturbance of rhythm which can be prevented to an appreciable extent, is auricular flutter. This may begin during the operation. More often it starts one or two days later. It is characterized by a racing rapid pulse (150 to 160 per minute) often attended by anxiety on the part of the patient and ending in exhaustion. Death may follow from cardiac failure. This complication can to a great extent be prevented by the routine administration of quinidine, one dose of which is given parenterally one hour before the anesthesia is started sometimes once during the procedure and daily thereafter for four or five days. It is given at first intramuscularly and then by mouth when the patient begins to take enough fluid orally.

MISCELLANEOUS COMPLICATIONS *Cerebral hemorrhages* and *thrombosis of the leg veins* are vascular complications which are no different after esophagectomy than after any other major procedure. The question of prophylaxis and treatment of phlebothrombosis by femoral vein ligation or the administration of anticoagulants like Dicumarol is one which must be settled in each instance according to the experience and beliefs of the surgeon in charge.

Other miscellaneous complications which bear no direct relation to esophagectomy as a specific procedure do not require mention.

Operative Mortality

The mortality rate after esophagectomy depends upon the relative complexity of the operation, the severity of the disease and the age and condition of the patient. With regard to the type of procedure the lowest mortality follows the Wookey operation and the highest the operations done for the removal of carcinoma in the superior mediastinal segment. The table (Fig. 394) summarizes the figures in the experience of one of the authors (R. H. S.).

Figure 395 shows the correlation between the age of the patient and the operative death rate. It is to be noted once again that the relative frequency of

POSTOPERATIVE MORTALITY FOLLOWING ESOPHAGECTOMY FOR CARCINOMA
OF THE ESOPHAGUS

Segment	Level of Anastomosis	Per cent
Cervical	Wooley (neck)	3
Superior mediastinal	Neck	50
Midthoracic	Supra aortic	25
Low thoracic	Infra aortic	7

FIGURE 394 Table showing postoperative mortality after esophagectomy for carcinoma at various levels

the disease increases steadily from one advancing age group to the next and that, as would be expected, the mortality percentage increases as well

There is reason for comment about these statistics. An operative mortality of 10 per cent or less after a procedure of considerable magnitude is obviously acceptable. Furthermore, there is not much objection to a death rate of 25 per cent in cases of midthoracic esophageal carcinoma when the hopeless prognosis of the disease is taken into account and the misery of the patient who goes unrelieved is considered. An operative mortality of 50 per cent, however, even with these considerations in mind, should be carefully reviewed. Because of the relatively small number of patients with carcinoma of the superior mediastinal segment and the few who are operated upon, this high figure may be a reflection of chance and not statistically significant. The technical difficulties involved, the stormy convalescence of many of the patients, and the unlikelihood that, with the exception of a few unusually favorable tumors, the disease can be eradicated give ample justification for the opinion that with carcinoma in the superior mediastinal segment surgical extirpation should be reserved for early favorable

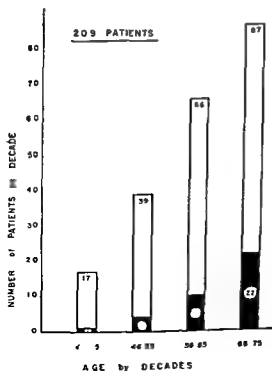


FIGURE 395 Chart showing the incidence of occurrence in numbers and postoperative mortality in percentage of patients operated upon at various ages for carcinoma of the esophagus (all levels)

tumors in younger patients who are relatively good risks for the operation. It is probable that the majority of patients with carcinoma in this area should be turned over to the radiologist for treatment.

End Results of Surgical Treatment

Before discussing the end results according to the experience of one of the authors (R H S) it should be stated that it is only the patients who survive the operation who should be considered in any evaluation of an operation for cancer. Those who died in the immediate postoperative period obviously could not demonstrate the outcome. All the charts to be presented, therefore, are based upon the numbers of patients who lived to prove what the result of the operation might be.

With the twofold therapeutic objective in mind of relieving many and curing some, it is interesting to observe what the results of surgical treatment are. The length of survival naturally depends upon the possibility of performing a satisfactory cancer operation. As discussed above, the success of the procedure from this point of view increases generally from above downward, with the least favorable results in the superior mediastinal segment and the best results in the lower segment. The technical reasons for this and their anatomical basis have already been discussed.

It should not be forgotten also that with a group of patients who are predominantly elderly, some actually in advanced age, the life span is so limited that many of those who die during the first months or years after the operation succumb to cardiovascular or other disorders characteristic of old age.

Furthermore, it should be mentioned that the survival from the point of view of so called cancer cure in any group of patients depends upon the policy employed with regard to the use of the operation. With carcinoma of the esophagus perhaps more than in any other region of the body, excepting possibly the rectum, the emphasis should be upon providing means of relief from the miseries of the disease. With this in mind it is necessary to offer surgical extirpation to all patients who do not have some serious contraindication or evidences of inoperability. The presence of distant metastases falls into this category.

In evaluating the results in any group, therefore, which has been chosen on the above basis, the outcome may appear on first thought to be rather disappointing because the series is so heavily weighted with what are obviously incurable cases from the first. This is clearly shown in the charts to be presented.

Carcinoma of the Cervical Segment

Because the disease in the experience of the author is not common in this region and because the policy of operating only in the early favorable cases, which was adopted after a short trial with some of the advanced tumors suggested this as a better course, the total experience of carcinoma in this region is small. The statistics therefore are not mathematically very significant. The results are summarized in Figure 396.

From this it is apparent that of thirty-two patients seen, only eleven (approximately 33.3 per cent) had a resectable lesion according to the criteria out-

RESULTS OF SURGICAL TREATMENT OF CARCINOMA OF THE CERVICAL SEGMENT

Resection attempted		21
Inoperable (exploration only)	10	
Wookey operation	11	
Alive with disease 3 years	1	
Alive without disease		
5 or more years	2	
Operation not advised		$\frac{11}{32}$
TOTAL		32

FIGURE 396 Table showing results of treatment of carcinoma of the cervical segment, lined above Two of these patients have survived over five years without evidence of disease, a suggestive five year survival of 20 per cent

Carcinoma of the Superior Mediastinal Segment

Fortunately carcinoma in this most unfavorable segment of the esophagus is very unusual Furthermore, as mentioned before, the resectability is relatively low Of the small number of patients in whom a resection was performed, none survived as long as five years

Carcinoma of the Midthoracic Segment

These are the cases which require the performance of an intrathoracic anastomosis above the level of the aortic arch Because of the fact that at the time of the operation a large number of the patients had obvious evidence of metastases usually to lymph nodes and in many instances definite spread beyond the

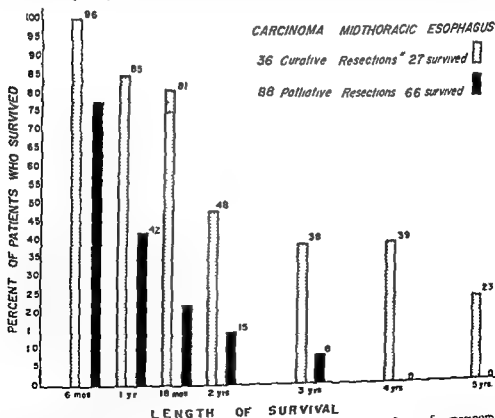


FIGURE 397 Chart showing the five year survival of patients operated upon for carcinoma of the midthoracic portion of the esophagus grouped according to whether favorable with a good prognosis, or strictly palliative

scope of the resection performed, it is proper to subdivide the group as a whole into (1) those with a favorable prognosis and (2) those in whom the operation was obviously only palliative

The first group consists of patients whose tumor was small enough to be removed along with a large margin of normal tissue and who had no obvious lymph node metastases with the exception of an occasional positive node on the excised specimen. The second group includes all those in whom nodal or other metastatic involvement was known to be left behind or whose tumor had penetrated the wall of the esophagus or was accompanied by numerous positive nodes. These are the cases where nothing but palliation was expected.

Figure 397 is a chart based upon the experience of 124 patients operated upon. It will be seen from this that of those who survived after the removal of a favorable growth according to the above criteria (so called curative resection), 23 per cent were alive five or more years after the operation. In the purely palliative cases 8 per cent of the patients were alive after three years, but none lived as long as five years.

Carcinoma of the Lower Esophagus and Cardia

As might be expected the survival statistics are best of all for this group. With these patients, as with those of the preceding group, it is important to make a subdivision according to whether the growth was favorable and the

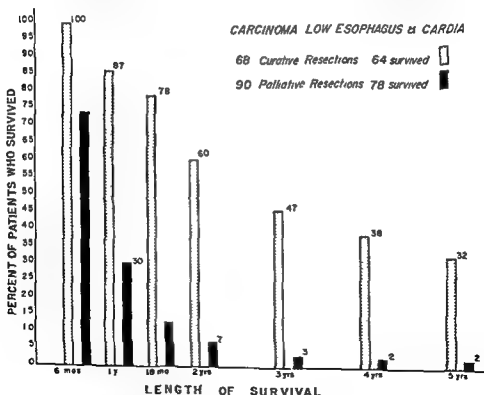


FIGURE 398 Chart showing the five year survival of patients operated upon for carcinoma of the lower esophagus and cardia grouped as to whether favorable or palliative. Note: Subdivision according to histologic type (epithelioma or adenocarcinoma) shows no survival difference.

operation therefore possibly "curative" or whether the operation was purely palliative (Fig 398). Here of the 142 patients who survived the operation, out of 158 operated upon, the five year survival in the favorable group ("curative resections") was 32 per cent. An interesting fact is that, of the patients whose operation was considered purely palliative, 2 per cent survived five or more years, which suggests that it is not always possible on clinical grounds alone to make a reliable prediction about the outlook for survival in a given patient. This lends further support to the radical policy of attempting to remove the growth in every patient when it appears to be possible.

In the United States there is currently a feeling of discouragement among surgeons with the results of surgical treatment. Many have reported statistics which are much less favorable than those of the personal experience just reviewed. These results are probably either the outcome of too small an experience with not enough cases to include a proportion of more favorable lesions, or the consequence of inadequacies of the removal of the tumor and the regional lymph nodes.

Palliation

The above are the long-term survival statistics, but the other side of the coin should be viewed as well. The relief which the operation provides is long lasting and in every respect superior to that provided by other methods. In fact, with the exception of carcinoma of the cervical segment, which is a case unto itself, it is a rare experience for any of the patients who succumb after esophagectomy to die with recurrent dysphagia. In the majority of instances, if they do not succumb to some intercurrent disease, they die of distant metastases still able to swallow until the end.

Surgical Methods for Palliation

As has been emphasized already, by far the most successful method for securing lasting relief from the dysphagia which makes the patient with a carcinoma of the esophagus so much an object of pity is a wide excision of the tumor followed by immediate restoration of continuity of the alimentary canal by esophagogastric anastomosis. Other methods may be mentioned as follows:

GASTROSTOMY The performance of a gastrostomy on a patient suffering from total esophageal obstruction caused by an inoperable carcinoma is often not a kindness. It does not overcome the distressing symptom of dysphagia. The most that can be said for it is that it makes possible the avoidance of the dehydration and starvation which the presence of the tumor would otherwise impose and prolongs a life of misery more humanely than can be accomplished by means of intravenous infusions of fluid electrolytes. It should rarely be performed and then only after every effort has been made to improve the ability of the patient to swallow, if only liquids.

USE OF AN INTRALUMINAL TUBE Several variations of this method have been employed. The most universally accepted is the insertion of the tube devised by Souttar (Fig 399). This consists of a flexible metallic tube of noncorrodable metal fitted with a flange at the upper end. The tube is introduced with a special instrument through an esophagoscope. An esophagoscope devised especially for this purpose by Negus facilitates its introduction.

The lumen through the tumor may require preliminary dilatation. A tube having a diameter of 18 to 20 mm. may then be inserted. Figure 400 shows a Souttar tube in the correct position. Such a tube permits the passage of liquid or even semisolid food and, provided it remains in position, gives the patient a considerable degree of relief, thereby avoiding the need for a gastrostomy.

There are two objections to its use. One is that in an appreciable number of patients the tube sooner or later slips from its lodging place in the growth and passes on into the stomach. This occurrence may coincide with the subsidence of associated inflammatory swelling or with an enlargement of the caliber of the lumen caused by the necrosis and sloughing of the central core of the tumor. The second is that it may ulcerate through the tumor or that it may take an incorrect course during its introduction and end up in the mediastinum. The appearance after such an accident is shown in Figure 401.

Modifications of this technique using tubes made of plastic materials have been described.

BOUGIENAGE in an effort to maintain a passageway through the tumor is rarely successful in achieving its objective and exceedingly dangerous because of the ease with which a perforation may be induced. Its chief value is as an adjunct to radiation treatment.

LOCAL EXCISION OF THE TUMOR WITH INTERPOSITION OF A PLASTIC TUBE. This method first publicized by Berman, or one of several modifications, has had a limited vogue. It consists in the local removal of the disease-containing segment through a thoracotomy incision and the insertion of a plastic tube having a diameter just smaller than that of the esophagus to bridge the defect. The

FIGURE 399 Photograph of a Souttar tube



E. M. Negus

FIGURE 400 Roentgenogram of a patient with a Souttar tube in place through the lumen of a carcinoma of the lower esophagus (Courtesy of Dr. Negus)





FIGURE 401 *A* Roentgenogram of a patient with a Souttar tube the end of which has perforated the esophagus and is pointing posteriorly toward the spine (lateral view) *B* Esophagogram of the same patient showing extravasation of barium into the mediastinum

obvious impossibility of securing a permanently tight connection between the ends of this foreign body and the living tissues of the esophagus is the chief fault of the method. The leakage which is so likely to occur at the point where the two are united carries the bacterially contaminated fluid from the esophagus into the mediastinum and often the pleural cavity. The large number of reports of mediastinal abscess and empyema following the use of this method of palliation has provided a dramatic clinical demonstration of the failure of a technique which, because it violates the basic principles of surgery and wound healing, could not be expected to result any differently. The method is mentioned only for the purpose of condemning it.

ESOPHAGEAL BY-PASS AND IRRADIATION TREATMENT In certain instances where there is a tightly obstructed growth which is unfavorable for resection, in a patient whose condition is still good it may be advisable to resort to the performance of a by-passing procedure followed by a course of intensive irradiation treatment. Although the stomach and jejunum can be utilized for this purpose the right colon provides a more simple and physiologically better means of accomplishing the objective. For the reasons explained in Chapter 20, where the technique is described, the anastomosis should be made in the neck after drawing the transplanted segment up through the anterior mediastinum.

Following this procedure the patient is relieved of the necessity of attempting to swallow through a stenosed esophagus and the roentgen treatment may be started within ten to fourteen days. With this arrangement, also the treatment is better tolerated and adequate nutrition of the patient can be maintained while it is being administered (see Chapters 20 and 29).

Irradiation Treatment of Carcinoma of the Esophagus

ALTHOUGH they have been employed for upward of thirty years, x-ray and radium therapy have not yet achieved much more than temporary amelioration of the disease. Recent technical improvements, however, give reason to hope for better results.

The difficulties of irradiation treatment for carcinoma of the esophagus can be attributed to several causes as follows:

1 *The rapidity with which lymphatic spread occurs* because of the extremely dense surrounding network of lymphatic channels which facilitates the process. In fact, the dissemination always takes place earlier than one would hope.

2 *The thinness and relative mobility of the esophagus* and in particular the absence of an impervious surrounding sheath of fatty cellular tissue which might provide a local barrier.

3 *The proximity of important organs* which lie in the field of irradiation (trachea, bronchi, lungs, heart, and aorta) and which should not be injured.

4 *The depth of the esophagus from the surface* which limits the effect of the irradiation and makes it difficult to appreciate the degree of extension with any accuracy.

Although many authors still consider that carcinoma of the esophagus is radioresistant, this opinion is debatable. The tumor is epitheliomatous and histologically falls into the category of lesions elsewhere which are known to respond favorably to x-rays. There is no clear-cut difference between the responses of the two histological types if comparable doses are given. There is practically no difference in radiosensitivity between these carcinomata arising in the esophagus and those which develop in other comparable types of mucous or mucocutaneous membranes. The radioresistance attributed to esophageal carcinoma is only apparent and is actually the consequence of inadequate irradiation.



FIGURE 401 A, Roentgenogram of a patient with a Souttar tube the end of which has perforated the esophagus and is pointing posteriorly toward the spine (lateral view) B Esophagogram of the same patient showing extravasation of barium into the mediastinum

obvious impossibility of securing a permanently tight connection between the ends of this foreign body and the living tissues of the esophagus is the chief fault of the method. The leakage which is so likely to occur at the point where the two are united carries the bacterially contaminated fluid from the esophagus into the mediastinum and often the pleural cavity. The large number of reports of mediastinal abscess and empyema following the use of this method of palliation has provided a dramatic clinical demonstration of the failure of a technique which, because it violates the basic principles of surgery and wound healing could not be expected to result any differently. The method is mentioned only for the purpose of condemning it.

ESOPHAGEAL BY-PASS AND IRRADIATION TREATMENT In certain instances where there is a tightly obstructed growth which is unfavorable for resection in a patient whose condition is still good it may be advisable to resort to the performance of a by-passing procedure followed by a course of intensive irradiation treatment. Although the stomach and jejunum can be utilized for this purpose the right colon provides a more simple and physiologically better means of accomplishing the objective. For the reasons explained in Chapter 20, where the technique is described, the anastomosis should be made in the neck after drawing the transplanted segment up through the anterior mediastinum.

Following this procedure the patient is relieved of the necessity of attempting to swallow through a stenosed esophagus and the roentgen treatment may be started within ten to fourteen days. With this arrangement, also the treatment is better tolerated and adequate nutrition of the patient can be maintained while it is being administered (see Chapters 20 and 29).

CHAPTER 29

Irradiation Treatment of Carcinoma of the Esophagus

ALTHOUGH they have been employed for upward of thirty years, x ray and radium therapy have not yet achieved much more than temporary amelioration of the disease. Recent technical improvements, however, give reason to hope for better results.

The difficulties of irradiation treatment for carcinoma of the esophagus can be attributed to several causes as follows:

1 *The rapidity with which lymphatic spread occurs* because of the extremely dense surrounding network of lymphatic channels which facilitates the process. In fact the dissemination always takes place earlier than one would hope.

2 *The thinness and relative mobility of the esophagus* and in particular the absence of an impervious surrounding sheath of fatty cellular tissue which might provide a local barrier.

3 *The proximity of important organs* which lie in the field of irradiation (trachea, bronchi, lungs, heart and aorta) and which should not be injured.

4 *The depth of the esophagus from the surface* which limits the effect of the irradiation and makes it difficult to appreciate the degree of extension with any accuracy.

Although many authors still consider that carcinoma of the esophagus is radioresistant this opinion is debatable. The tumor is epitheliomatous and histologically falls into the category of lesions elsewhere which are known to respond favorably to x rays. There is no clear-cut difference between the responses of the two histological types if comparable doses are given. There is practically no difference in radiosensitivity between these carcinomata arising in the esophagus and those which develop in other comparable types of mucous or mucocutaneous membranes. The radioresistance attributed to esophageal carcinoma is only apparent and is actually the consequence of inadequate irradiation.

tion The myth of the radioresistance of comparable carcinomata in the rectum has been destroyed now that it is known that the failures were the consequence of underdosage The same is probably true for carcinoma of the esophagus

Radium Treatment (Curietherapy)

Many authors have been or still are in favor of treating esophageal carcinomata with radium only First employed in 1904 by Exner of Vienna, later by Einhorn of New York and in France in 1909 by Guisez and Barcat, curietherapy was used subsequently by Hill, Guisez, Watson and Williams, Finzi, and Seucert who in his *Treatise on Diseases of the Esophagus and Stomach* expressed some reserve because of certain unfortunate experiences Following these pioneers the method was given further trial by others with modifications of technique including insertion of radon needles and seeds in addition to the conventional methods

METHODS OF APPLICATION For the treatment of carcinoma of the esophagus radium is generally applied internally (*endocurietherapy*), but it is possible also to use it by external percutaneous methods or through the neck after surgical intervention Certain authors have given irradiation from this source at a considerable distance from the skin and in large dosage This is spoken of as *telecurietherapy*

PRELIMINARY PREPARATION The esophageal lumen is a conduit into which are introduced, often forcibly, many objects capable of aggravating the inflammatory reaction and esophagospasm which usually accompany neoplastic lesions This should prompt those in charge of the patient to postpone the introduction of the radium-containing apparatus until an effort is made to improve the local situation If there is not too much constriction, the patient's condition is bolstered as much as possible by a good dietary regimen Dysphagia due to spasm is combatted by relaxing agents and antispasmodic medicaments

Many radium therapists do not carry out this preliminary preparation, but they commit a regrettable omission as well as a psychological error because by these measures the patient is given confidence and the introduction of the radium is made easier

Endocurietherapy

The application of radium or radon can be practiced either with needles and seeds or more usually with tubes containing the radioactive substance placed in an applicator which is introduced whenever possible into the aperture of the lumen through the tumor This may be accomplished either from above down or from below up by retrograde insertion The tubes may be placed end to end or separated by a slight interval in the interior of a soft sound and inserted so that the first tube extends slightly beyond the inferior margin of the tumor and the last rises somewhat above the superior margin (Fig. 402)

This demands an accurate localization of the limits of the growth which may be facilitated particularly when there is doubt about the extent along the esophagus by the combined method of visualization from above and below illustrated in Figure 403 This technique, however, can be applied only when

the patient has a gastrostomy or is able to swallow enough barium mixture to fill the stomach partially. Figure 402 shows the results which may be obtained by this technique.

The caliber of the sound should be of correct size to permit its passage through the tumor without difficulty. The positioning of the applicator is made with the aid of the usual controls including measurements from the dental arch and verification of the position by careful fluoroscopic observation. The importance of not subjecting normally healthy zones of mucosa to the caustic effect of large doses of irradiation must be realized.

When the aperture is not large enough to permit the passage of a sound the interior diameter of which is large enough to accommodate the tubes intraluminal application is impossible. Certain authors, however, advise the use of dilators on a continuous thread (see Chapter 19) to make the insertion possible.

In difficult cases when the danger of creating a false passage is feared the placing of the applicator may be accomplished by esophagoscopy. When it becomes necessary from time to time to remove and replace the applicator blind introduction with or without fluoroscopic control may be sufficient but it is necessary to have a definite means of making certain of the position of the radium which the patient or the attendants charged with his care may understand.

PROCEDURE USING THE ESOPHAGOSCOPE. In the method using a rubber applicator containing two or three tubes of radium in tandem, the number de-

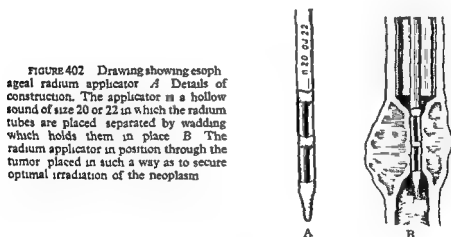


FIGURE 402 Drawing showing esophageal radium applicator. A Details of construction. The applicator is a hollow sound of size 20 or 22 in which the radium tubes are placed separated by wadding which holds them in place. B The radium applicator in position through the tumor placed in such a way as to secure optimal irradiation of the neoplasm.

FIGURE 403 Diagram showing method of visualizing both ends of a carcinoma by double ingestion of barium. The patient is placed in the Trendelenburg position and with the cardia open the barium in the stomach flows back up the esophagus to the lower border of the growth. Barium descending from above outlines the upper border. (Method of Ledoux and Sluys after Ducuing.)

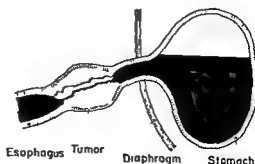




FIGURE 404 · Roentgenogram of a carcinoma the upper and lower margins of which have been defined by the method illustrated in Figure 403

pending on the length of the tumor, the introduction is made under esophagosopic control. After wiping out the stenosed lumen with pledgets soaked in a cocaine-Adrenalin solution, the applicator is put in position under visual control using a strong forceps. The forceps is then withdrawn, followed by the esophagoscope, without displacing the withdrawal string which is attached to the patient's cheek. The string can also be passed out through the nose by way of the nasopharynx.

Chevalier Jackson has a modification in which the retaining thread attached at one end to the radium applicator is threaded through the esophageal aspirator tube. By pulling on the thread the applicator is drawn up against the aspirator tube so that both are held rigidly together and can be introduced

through the esophagoscope. When the applicator has been put in place in the center of the tumor, the thread is detached, the aspirator is withdrawn, and then the esophagoscope.

Implantation of radium or radon-containing needles or seeds through the esophagoscope has been employed, but the method is blind and inexact because of the fact that the lower portion of the tumor may not be reached (Figs. 405 and 406). This method is rarely used at present.

APPLICATION WITHOUT ESOPHAGOSCOPY Many procedures have been devised, but they may be grouped under two headings:

1 *Flexible Applicators Method of Gunze* Two tubes of 50 mg of radium bromide are placed in tandem end to end in a position to irradiate the tumor. A silver or platinum shield is used on the applicator and there is, of course, the thickness of the containing rubber sound. The tubes are placed in the rubber sound.

The exact location of the stenosis is determined and its distance from the teeth is marked on the sound by tying on a thread. The sound is introduced under local anesthesia and put in correct position. The outer end is then fastened with a thread around the neck or head.

2 *Semirigid Applicators Method of Sargnon* Sargnon has employed the same technique in many cases but he prefers whenever possible the application of radium after gastrostomy to provide a means of feeding the patient.

Introduction with the aid of a thread in the esophagus may be used. Two types of cases must be considered depending on whether or not the patient has a gastrostomy. If there is a gastrostomy, the patient is made to swallow a string which is subsequently drawn out through the gastrostomy stoma as was de-



FIGURE 405. Roentgen film showing radon needles implanted in a carcinoma of the upper mediastinal segment of the esophagus (Courtesy of Dr. Negus).



FIGURE 406 . Roentgenogram showing radon seeds implanted in a carcinoma of the mid thoracic segment of the esophagus (Courtesy of Dr. Negus)

scribed in Chapter 19 and the lower end tied to the radium applicator. The introduction is then made from below upward by pulling on the thread.

In a person who does not have a gastrostomy, a thread is swallowed by the patient at a rate of about 30 cm. an hour until it becomes fixed in the intestine. This fact is made known when there is considerable resistance to traction on the outer end. The applicator is then passed down the thread to the proper position in the same manner as a dilator would be used on a thread.

Another method is to incorporate the radium applicator in the end of a nasogastric rubber tube or sound which is introduced through the nose as a Levin tube might be inserted (Fig. 407).

The application of radium if the patient has not had a previous gastrostomy makes the taking of food and liquids impossible while the apparatus is in place. A sound with grooves on the outside has been devised to make it possible to get a little liquid down, or one may use a tube with the radium placed around its circumference. These procedures, however, can only be carried out under esophagoscopic control in a stenosis of large diameter and after preliminary dilatation. They are therefore impractical in the long run.

Whatever may be the method employed, fluoroscopic verification of the correctness of the position of the radium-containing apparatus and of the possibilities of its getting out of position is essential. If the application does not

succeed in affecting the entire length of the tumor, the tube must be introduced a little further during the treatment in order to irradiate the lower portion of the neoplasm

DOSAGE The dosage depends on whether only one or a series of applications is used. With a single application it is necessary in order to obtain the maximum effect to use at least 50 mg of radium bromide held in place for twenty-two to twenty-three hours. At the London Radium Institute the doses vary from 70 to 100 mg for eighteen to twenty-four hours which gives about 5 to 10 millicuries to the growth. Others have used as much as 16 to 18 millicuries. A second application may still be given at a later date but with some misgivings because of the weak resistance of the tissues already heavily irradiated.

When a series of applications is given only a short interval should be allowed between treatments. Innumerable variations of technique are possible. The dosage should depend upon the bulk and extent of the tumor. For an average tumor 4 to 5 cm long the Ducuing brothers employ three tubes, each containing 5 mg of radium element. The total duration of treatment is 260 to 300 hours partitioned over a period of fifteen to twenty days with a resulting dosage of 30 to 32 millicuries. Whenever possible they prefer curietherapy using a sound by the method of Guisez.

Certain points should be emphasized (1) An early diagnosis is essential (2) In the hope of securing a good result only localized lesions or those apparently confined to the wall of the esophagus should be treated (3) The exact location of the carcinoma in relation to the upper dental arch must be measured in order to estimate correctly the length of the radium containing apparatus

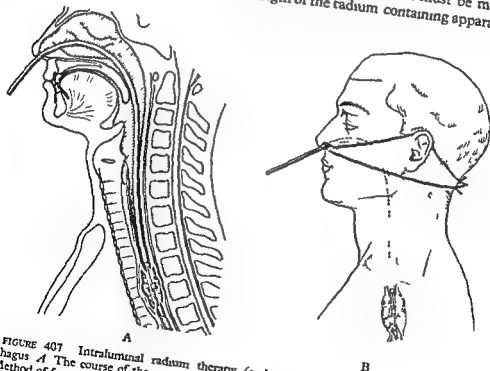


FIGURE 407 Intraluminal radium therapy (endocurietherapy) of carcinoma of the esophagus. *A* The course of the sound and position of the radium applicator in the tumor. *B* Method of fastening the apparatus (Sargnon technique).

needed Fluoroscopy permits one to obtain a good idea of the length of the stenosis and consequently of the tumor to be irradiated

CONTRAINDICATIONS : Contraindications arise from the narrowness of the esophageal opening and its nondilatability, the spread of the tumor to neighboring organs or tissues, distant metastases, lymph node involvement, laryngeal paralysis, the presence of a perforation or fistula, poor general condition, fever, or deterioration of the intellectual faculties

COMPLICATIONS : Complications are not rare and are always serious The number of cases has not been publicized In general during the period of the application and the first few days thereafter, there are few or no complications Fatal hemorrhage, however, has been noted

During the weeks which follow the application, too much destruction of the wall of the esophagus may result in perforation or even the formation of a fistula Pleural and pulmonary complications may develop because of intense action of the irradiation Perforation of the aorta may occur for the same reason Gangrene of the esophagus has been reported

Especially in emaciated cachectic patients one may observe shock, generalized toxicity from rapid absorption of septic products, and pulmonary infections, often as a result of fistula formation

Other complications are false passages, mediastinitis, phlegmons of the mediastinum, or in the rare case where a favorable outcome has been observed, a cicatricial stenosis Dilatation may be necessary in the latter instance

Telecurietherapy

To make the rays homogeneous it is necessary to lengthen the distance between the radiation source and the patient This requires more powerful units The technique is called "telecurietherapy" The quantity of radium required varies from 4 to 10 grams The method is, however, not economical and can be employed only in certain centers which possess large quantities of the element Furthermore, it does not possess any advantages over roentgen therapy and the results are no better The latter technique is preferable, therefore, unless the use of radium may eventually be shown to be superior Actually the principles of usage of the two methods are the same It is the radiation source only which is different

Results of Radium Therapy

It is not surprising that results have been disappointing even when compared with those following roentgen therapy The effect is inevitably sharply localized to the primary tumor Lymph node metastases even when the nodes are close to the tumor site are beyond the effective range of the emanation which, practically speaking, affects only the growth itself From the practical point of view, therefore, little more than temporary palliation can be expected in the average case of carcinoma of the esophagus With these factors in mind, it is easy to understand why radium therapy has been almost universally abandoned as a method of treating this disease

Treatment by Roentgen Irradiation

Until fifteen years ago, roentgen therapy of carcinoma of the esophagus was considered merely palliative and there was little or no enthusiasm for its use. Since then two facts have intervened to produce a revival of interest in this method. In the first place, the development of thoracic surgery has made it possible to remove and even to cure a certain number of these growths. In the second place, technical progress and improvements in equipment for roentgen therapy have aroused the roentgenologists from their lethargy and forced them to re-evaluate the treatment of this supposedly incurable disease and to compare their statistics with those of the surgeons.

Progress began with improvements of the apparatus, as a result of which better depth deliveries are obtained by the utilization of higher voltages (now 2 000,000 volts) which permit a greater degree of penetration and an increased focal distance. In addition, thanks to an extremely accurate irradiation technique by the method either of cross-firing or of rotation therapy, the rays can be directed more effectively. Finally, there has been developed a better means of calculating the doses actually delivered to the tumor.

Calculation of Dosages

All roentgen therapy is based upon the administration of a sufficient dose of x rays to the tumor itself. This dose, called the *lethal dose*, should be 5000 to 6000 roentgen units (r). It is obtained by the conjunction of different points of technique which will be presented. Nothing but a glimpse of these different problems, the details of which are to be found in the books devoted to the subject, will be given here.

Depth delivery has been studied by numerous authorities. It is presented in tables which are available for use. That developed by Paterson is a convenient type. One can calculate as a general rule that the depth delivery or tumor dose is approximately one-fifth of the dose delivered at the skin. It is necessary therefore to give large doses which present considerable risk of causing serious skin damage. To calculate the correct dose in a given patient it is necessary to consider not only the quality of the irradiation employed but also the field to be treated and the interval between the irradiation treatments.

1. The QUALITY (CHARACTERISTICS) OF THE IRRADIATION is determined by measuring the effect of the apparatus on test objects. According to Lefèvre, the ideal for this measurement is to be able to introduce a small ionization chamber into the esophagus in contact with the tumor. This would make possible an exact determination of the dose delivered. Paterson makes a mold of the thorax of the subject, localizes the situation of the tumor and calculates the distance between the lesion and the skin to determine the proper dose to administer.

The apparatus must be sufficiently powerful to function at 180 kv. at least. Technical progress has evolved machines which deliver 200, 400 and 800 kv., all of which seem to produce about the same results. Since the availability of the 1,000,000 and more recently 2,000,000 volt apparatus, the effect is definitely superior in that a greater degree of filtration and a longer focal distance are possible. Not all hospitals and x-ray laboratories are equipped with such high

voltage machines. Many workers therefore employ on the average a kilovoltage of 200 kv with a filtration of 1 mm of copper plus 2 mm of aluminum and a focal distance of 50 to 60 cm. In this way a sufficient degree of penetration of the roentgen rays is provided to obtain satisfactory results.

2. The FIELD OF IRRADIATION, as for all deep radiotherapy, bears a direct relation to the skin reaction. It is necessary to deliver to the tumor a dose larger than that which the skin receives. This is the reason for the development of the "cross-fire" technique. Successive improvements have evolved three methods of application using this principle.

The *classical technique* employs four fields: two anterior parasternal, one right and the other left, and two posterior paravertebral, also right and left. The fields are of rather large size (6 to 8 cm by 15 cm on the average). With the usual lower voltage apparatus it is difficult to give more than 3000 r over each field without provoking serious cutaneous damage. The total external dose therefore will be in the vicinity of 12,000 r and the depth dose approximately 2500 r. This treatment can hardly be curative and this method is actually now reserved for patients requiring palliation only, as we shall see further on. The centering is the same as for other methods which will be described more precisely. The use of four fields was for a long time the only technique employed by radiologists and is the one on which the opinion that carcinoma of the esophagus is not curable by x-ray was based. Nowadays it can be shown by using other techniques that the tumor can be destroyed. Two such methods have been evolved.

The *multiple field method of Paterson* consists in increasing the number of portals to produce a tumor dose of 6000 r. To be effective, this method demands a very accurate aim upon the lesion to be irradiated. To accomplish this by the technique devised by Paterson, a plaster jacket is molded around the patient. On this jacket the localization of the limits of the tumor are indicated as disclosed by fluoroscopic examination. Thanks to a system of comparisons, the portals of entry and exit of the rays are delineated on this jacket. It is then very simple, by means of a viewer adapted to the roentgen therapy apparatus, to reposition the patient in the same angles of incidence. In addition, wax markers are attached to the jacket. Thus it is easy for an operator to center the patient with the least chance of error.

Paterson irradiates from six to twelve fields, which obviously demands great accuracy in centering the patient. These fields are 6 cm by 12 to 15 cm each one almost touching the other. The height of the fields corresponds to twice the apparent length of the tumor (vertical dimension). The maximum is 20 cm or even more in certain extensive tumors. The dose to be given to each field varies and is calculated as a function of the exact distance from the skin to the tumor for each incidence in terms of the irradiation received through the opposite fields on the other side of the thorax. By thus modifying the portals of entry it is possible to give 6000 r to the tumor without too much effect upon the skin.

This extremely precise technique has been a little simplified by numerous authors who are usually satisfied with six to eight fields. They localize the placement of each field directly on the skin (without a plaster jacket) and it is the

physician, not a technician, who centers the patient beneath the roentgen therapy apparatus at each treatment

Rotation therapy as popularized by Jens Nielson The idea of rotational therapy was conceived by Kohl in 1908. It was put into practice in 1916 by Knox and Caufield. It was not until 1941, however, that Jens Nielson after trial published a number of articles setting forth the details of his technique. Mesnil de Rochemont published his work at about the same time.

The principle is merely a modification of the multiple field method, but the fields are multiplied to infinity by turning the patient or the x-ray tube in such a way that the axis of rotation corresponds to that of the tumor being irradiated. There is therefore a cylindrical field which girdles the patient at the level of the growth. The usual apparatus is at present a rotating chair in which the patient is seated (Fig. 408).

This method, which is very simple in principle, is in reality rather complicated to carry out because of the necessity of having the tumor exactly centered throughout the period of treatment. Nielson and others who have perfected this technique have the patient swallow some barium while seated on the rotating chair and use a fluoroscope with which to center the tumor in relation to the radiotherapy tube. This surveillance must be constant and requires the presence of the physician throughout the period of irradiation. He is protected in a lead enclosure and by a system of controls can regulate the diaphragm of the opening and the centering of the tube (Fig. 408).

Krebs, Howard, Nielson, and Anderson who published their results a few years ago consider that this fluoroscopic centering during irradiation is not ab-

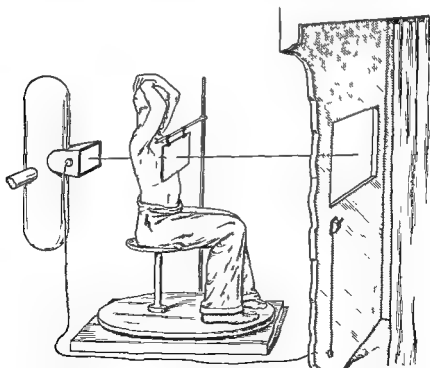


FIGURE 408 Diagram illustrating the method of rotation therapy using roentgen rays. Patient sits on a revolving seat.

olutely necessary. They fix the patient on his seat by means of plastic straps to prevent him from moving. They have previously marked out the projections of the tumor on the skin in two incidences at right angles to each other. They then make the two planes thus determined coincide with two other planes marked on the floor, which indicate the axis of rotation of the turning seat. They are well satisfied with their results and the method presents the advantage that it can be carried out by a single technician. The speed of rotation varies from one to fifteen minutes per complete turn.

The size of the field is 15 by 5 cm. on the average. The delivery percentage is from 300 to 350. With doses of 25,000 to 30,000 r at the skin, a tumor dose of 5000 to 6000 r is obtained. The cutaneous reaction is limited to a slight dry dermatitis.

3. FREQUENCY OF TREATMENTS. For the separate field techniques irradiation of one field per day, giving 150 to 300 r at each seance, is an average program until 3000 to 5000 r per field has been administered. Certain authorities sometimes irradiate two fields per day, giving half the dose to each. The total duration of treatment is four to eight weeks.

For rotation therapy Jens Nielson gives from 100 to 200 r per day in two sittings. The total duration is from five to six weeks.

Indications for Roentgen Therapy

In principle, all carcinomata may be benefited by x-ray treatment. The relative indications for roentgen therapy as opposed to surgical excision need not be discussed here. The purpose is merely to define the employment of the different methods of irradiation. Paterson is correct in making a distinction between treatment which is curative and that which is palliative. The first is undertaken with the objective of destroying a tumor which is still curable. The second attempts merely to ameliorate the symptoms presented by a patient whose disease has gone past the stage of curability.

RADICAL TREATMENT. Before starting a course of treatment with intent to cure the disease, it is essential to establish two important points in order to know whether the undertaking is reasonable.

First, the general condition of the patient should be investigated. He must be able to swallow soft foods easily. In fact, in order for a patient to be able to undergo an intensive course of x-ray treatments he must be able to nourish himself with a fair amount of ease in spite of the additional dysphagia due to the reactionary swelling of the esophageal tissues caused by the treatment. If it becomes necessary to perform a gastrostomy, the maintenance of nutrition is not so satisfactory. Furthermore, the operation has a depressing effect upon the morale of the patient which is an important consideration. The need for a gastrostomy is actually a contraindication to radical treatment.

In addition, the existence of mediastinitis, the evidences of invasion of mediastinal organs, and the discovery of metastases contraindicate radical treatment.

Secondly, the dimensions and possible extension of the lesion are important. If the tumor is over 5 cm. in length, there is a strong chance that there are already lymph node metastases. It is wise, therefore, to limit the use of

radical therapy to tumors under 5 cm in length. Other authors have set limits of 8 cm or even 10 cm.

An esophagoscopy should be performed not only to secure documentation by biopsy but also to help establish the exact limits of extension of the tumor.

It is important and interesting also to keep track of the regression of the lesion radiologically. Lefèvre calls attention to the fact that the segmental rigidity which appears first disappears last. In a successful case the pliability of the esophageal walls can be seen to return completely.

For radical treatment as mentioned above, a dose of 5000 to 6000 r must be given.

PALLIATIVE TREATMENT Treatment for palliation is indicated in patients whose tumor cannot be excised or who are not good candidates for the intensive treatment described above. Two categories of patients must be distinguished: (1) If the tumor measures over 5 cm and the patient is in good general condition, or under 5 cm and deglutition is impossible, or if the subject with a tumor less than 5 cm in length is aged or in too poor a condition to withstand intensive treatment, one should be satisfied with a dosage of 3000 to 4000 r given in four to eight days in order to ameliorate the symptoms by retarding the growth of the tumor for a time. (2) If the tumor is extensive if there are signs of mediastinitis, or an esophagobronchial or tracheal fistula, or metastases, and if the growth is located at the cardia and has invaded the stomach, a small dose of 2500 to 3000 r is sufficient merely in an effort to temporize.

COMBINED TREATMENT Intraluminal irradiation combined with irradiation from some external source, usually the roentgen ray machine, may have some advantages and has had a few advocates. The value of this technique is debatable, however, and the method is subject to the disadvantage that it subjects the patient to the additional discomforts and dangers of radium treatment while not offering much additional benefit.

Discomforts and Complications of Roentgen Therapy

Intensive treatment leads to all the manifestations of intolerance to x-rays which are well known and do not need re-emphasis. So far as the esophagus itself is concerned, there is often enough *edema and swelling* during the early phase of treatment to increase the dysphagia or even lead to complete obstruction if the encroachment on the lumen is already great. This tendency can to a large extent be prevented by the administration of Meticorten during the course of the treatments (see below).

After two or three weeks of treatment the patient may also experience considerable *soreness* which with a carcinoma at the upper end of the esophagus renders deglutition so painful that the patient may not be able to consume enough food for maintenance of nutrition even if the obstruction is not great. This occurrence, also, is eliminated or to a great extent controlled by giving Meticorten.

The degree of *cutaneous reaction* bears a direct relation to the voltage of the apparatus. It is greatest with multiple field low voltage treatment and least severe after rotational therapy with the 2 000,000 volt machines. As already mentioned, this is the principal advantage of the higher voltage techniques.

Mediastinal and pulmonary fibrosis are very frequent and may even lead to spots in the lung where the blood vessels and lymphatics are completely obliterated by the fibrous contraction. This tendency is also more marked with the multiple field method than with the rotation technique.

Cicatricial contraction of the tumor site as the neoplastic tissue gives way to the ingrowth of fibrous tissue is a frequent occurrence. In fact, with the majority of patients it is necessary to combine bougienage with the roentgen therapy. For this purpose, in all but the relatively unobstructed vegetative type of tumors, it is wise to cause the patient to swallow a thread before the treatment is started in anticipation of subsequent need for dilatation.

Whenever the growth is so advanced that it has actually invaded the wall of the trachea or a bronchus, the end result of treatment as the tumor is destroyed is almost invariably the creation of a fistula. In every patient, therefore, whose tumor is in the juxtatracheal or bronchial segment, a tracheobronchoscopy must be performed, and if the growth is found to be invading the air passage, roentgen treatment should not be given.

Invasion of the wall of the aorta often ends in a fatal hemorrhage as the growth is destroyed by the roentgen rays. For this reason, unusually large tumors in the portion of the esophagus which lies next to the aorta should not be given large doses.

Adjuvant Treatment

Medical treatment for the skin reaction needs no comment.

The maintenance of nutrition during the treatment is of paramount importance. Swallowing is often difficult either because of swelling or because of pain. As mentioned above, both of these discomforts are largely eliminated by the exhibition of Meticorten, 5 mg. every six hours (or four times a day) during the course of treatment. The same precautions must be observed as for any utilization of this steroid hormonal substance.

One must provide a suitable dietary formula which the patient may be able to swallow. That devised by Paterson is as follows:

Milk	1000 cc
Butter	40 gm
Flour	20 gm
Sugar	30 gm

These ingredients are mixed and homogenized in a blender. To this is added the yolk of one egg. A small amount of brandy or rum may be added to give it flavor. Vitamins may be added to the mixture or given parenterally. If the feedings are given chilled, they are easier to swallow. The daily amount supplies approximately 2000 calories.

If the patient can swallow more than just liquids, custard, junket, gelatin, creamed soups, pureed vegetables, etc., may be added to the diet.

When the obstruction is complete, a *gastrostomy* must be performed before treatment is started. In borderline cases in which obstruction is imminent and when the patient is unable to swallow a string as a means of aiding the passage of a bougie, a gastrostomy is also indicated. The operation is to be avoided if possible, however, for obvious reasons.

Results of Roentgen Therapy

It is difficult to obtain any clear cut information about the results of roentgen therapy which can be compared with the large experience with surgical treatment now available. Obviously this is because the choice of patients for the two types of treatment is different. In the majority of instances surgery is given the first chance and roentgen therapy has been confined to those patients whose growths are apparently inoperable or who are too old or who have physical contraindications to surgical interference. No large series of patients treated with roentgen therapy by preference, which might be properly compared with a surgically treated group, is yet available. The most nearly comparable series so far as choice of cases is concerned is that of Buschke and Cantrell but it is small.

From the point of view of survival, the most that can be said is that, whereas formerly without any treatment 100 per cent of the patients died in less than two years after the diagnosis was made, with modern x-ray therapy the number who live beyond two years is considerable. Furthermore, there is an increasing proportion of patients, small to be sure, who survive five or more years.

Lefèvre reports 10 per cent survival for more than two years, without evidence of recurrence or metastasis.

Krebs and associates reported 500 cases of patients treated by the rotational technique. Of these 33.3 per cent died in less than three months, 50 per cent in less than one year, and 90 per cent in less than two years leaving 10 per cent who survived beyond two years.

Jens Nielson reports improved survival using the rotation technique as follows: Over one year 25 per cent, as compared with 10 per cent previously, and over two years 15 per cent, as compared with 4 per cent previously.

Many authors are able to report a few cases of survival for five or more years with an apparent preponderance in favor of the higher voltage techniques although the total numbers are too small to draw final conclusions. Fleming in a review of the literature collected 800 cases from various sources with ten survivals for five or more years.

So far as palliation is concerned the results are only fairly satisfactory and so much inferior to those of surgical resection using modern methods as to bear a most unfavorable comparison. The average experience of many workers in this field (Krebs and associates, Moulouguet, Lefèvre, Baclesse, Buschke and Cantrell etc.) is the disappearance of the tumor in 20 per cent of the patients treated. This is based upon roentgenological and esophagosopic evidence. It is interesting that in isolated instances in the experience of everyone who uses the method complete disappearance of the tumor has been observed at autopsy. Fried has reported three such cases. The metastases so frequently present, however, ultimately cause the death of these patients. Many of these 20 per cent of patients develop stenosis as a result of cicatrization and require additional treatment by bougienage.

Deglutition is definitely improved by elimination of the neoplastic stenosis in 4 per cent of the patients reported by Krebs and in a varying number as

Mediastinal and pulmonary fibrosis are very frequent and may even lead to spots in the lung where the blood vessels and lymphatics are completely obliterated by the fibrous contraction. This tendency is also more marked with the multiple field method than with the rotation technique.

Cicatricial contraction of the tumor site as the neoplastic tissue gives way to the ingrowth of fibrous tissue is a frequent occurrence. In fact, with the majority of patients it is necessary to combine bougienage with the roentgen therapy. For this purpose, in all but the relatively unobstructed vegetative type of tumors, it is wise to cause the patient to swallow a thread before the treatment is started in anticipation of subsequent need for dilatation.

Whenever the growth is so advanced that it has actually invaded the wall of the trachea or a bronchus, the end result of treatment as the tumor is destroyed is almost invariably the creation of a fistula. In every patient, therefore, whose tumor is in the juxtatracheal or bronchial segment, a tracheobronchoscopy must be performed, and if the growth is found to be invading the air passage, roentgen treatment should not be given.

Invasion of the wall of the aorta often ends in a fatal hemorrhage as the growth is destroyed by the roentgen rays. For this reason, unusually large tumors in the portion of the esophagus which lies next to the aorta should not be given large doses.

Adjuvant Treatment

Medical treatment for the skin reaction needs no comment.

The maintenance of nutrition during the treatment is of paramount importance. Swallowing is often difficult either because of swelling, or because of pain. As mentioned above, both of these discomforts are largely eliminated by the exhibition of Meticorten, 5 mg. every six hours (or four times a day) during the course of treatment. The same precautions must be observed as for any utilization of this steroid hormonal substance.

One must provide a suitable dietary formula which the patient may be able to swallow. That devised by Paterson is as follows:

Milk	1000 cc
Butter	40 gm
Flour	20 gm
Sugar	30 gm

These ingredients are mixed and homogenized in a blender. To this is added the yolk of one egg. A small amount of brandy or rum may be added to give it flavor. Vitamins may be added to the mixture or given parenterally. If the feedings are given chilled, they are easier to swallow. The daily amount supplies approximately 2000 calories.

If the patient can swallow more than just liquids, custard, junket, gelatin, creamed soups, pureed vegetables, etc., may be added to the diet.

When the obstruction is complete a gastrostomy must be performed before treatment is started. In borderline cases in which obstruction is imminent and when the patient is unable to swallow a string as a means of aiding the passage of a bougie, a gastrostomy is also indicated. The operation is to be avoided if possible, however, for obvious reasons.

Results of Roentgen Therapy

It is difficult to obtain any clear cut information about the results of roentgen therapy which can be compared with the large experience with surgical treatment now available. Obviously this is because the choice of patients for the two types of treatment is different. In the majority of instances surgery is given the first chance and roentgen therapy has been confined to those patients whose growths are apparently inoperable or who are too old or who have physical contraindications to surgical interference. No large series of patients treated with roentgen therapy by preference which might be properly compared with a surgically treated group, is yet available. The most nearly comparable series so far as choice of cases is concerned is that of Buschke and Cantril, but it is small.

From the point of view of survival, the most that can be said is that, whereas formerly without any treatment 100 per cent of the patients died in less than two years after the diagnosis was made, with modern x-ray therapy the number who live beyond two years is considerable. Furthermore, there is an increasing proportion of patients, small to be sure, who survive five or more years.

Lefèvre reports 10 per cent survival for more than two years without evidence of recurrence or metastasis.

Krebs and associates reported 500 cases of patients treated by the rotational technique. Of these 33.3 per cent died in less than three months, 50 per cent in less than one year, and 90 per cent in less than two years, leaving 10 per cent who survived beyond two years.

Jens Nielson reports improved survival using the rotation technique. It follows: Over one year 25 per cent, as compared with 10 per cent previously, and over two years 15 per cent as compared with 4 per cent previously.

Many authors are able to report a few cases of survival for five or more years with an apparent preponderance in favor of the higher voltage techniques although the total numbers are too small to draw final conclusions. Fleming in a review of the literature collected 800 cases from various sources with ten survivals for five or more years.

So far as palliation is concerned, the results are only fairly satisfactory and so much inferior to those of surgical resection using modern methods as to bear a most unfavorable comparison. The average experience of many workers in this field (Krebs and associates, Moulouguet, Lefèvre, Baclesse, Buschke and Cantril, etc.) is the disappearance of the tumor in 20 per cent of the patients treated. This is based upon roentgenological and esophagoscopy evidence. It is interesting that in isolated instances in the experience of everyone who uses the method complete disappearance of the tumor has been observed at autopsy. Fried has reported three such cases. The metastases so frequently present however ultimately cause the death of these patients. Many of these 20 per cent of patients develop stenosis as a result of cicatrization and require additional treatment by bougienage.

Deglutition is definitely improved by elimination of the neoplastic stenosis in 4 per cent of the patients reported by Krebs and in a varying number as

Mediastinal and pulmonary fibrosis are very frequent and may even lead to spots in the lung where the blood vessels and lymphatics are completely obliterated by the fibrous contraction. This tendency is also more marked with the multiple field method than with the rotation technique.

Cicatricial contraction of the tumor site as the neoplastic tissue gives way to the ingrowth of fibrous tissue is a frequent occurrence. In fact, with the majority of patients it is necessary to combine bougienage with the roentgen therapy. For this purpose, in all but the relatively unobstructed vegetative type of tumors, it is wise to cause the patient to swallow a thread before the treatment is started in anticipation of subsequent need for dilatation.

Whenever the growth is so advanced that it has actually invaded the wall of the trachea or a bronchus, the end result of treatment as the tumor is destroyed is almost invariably the creation of a fistula. In every patient, therefore, whose tumor is in the juxtatracheal or bronchial segment, a tracheobronchoscopy must be performed, and if the growth is found to be invading the air passage, roentgen treatment should not be given.

Invasion of the wall of the aorta often ends in a fatal hemorrhage as the growth is destroyed by the roentgen rays. For this reason, unusually large tumors in the portion of the esophagus which lies next to the aorta should not be given large doses.

Adjuvant Treatment

Medical treatment for the skin reaction needs no comment.

The *maintenance of nutrition* during the treatment is of paramount importance. Swallowing is often difficult either because of swelling or because of pain. As mentioned above, both of these discomforts are largely eliminated by the exhibition of *Meticorten*, 5 mg. every six hours (or four times a day) during the course of treatment. The same precautions must be observed as for any utilization of this steroid hormonal substance.

One must provide a suitable dietary formula which the patient may be able to swallow. That devised by Paterson is as follows:

Milk	1000 cc
Butter	40 gm
Flour	20 gm
Sugar	30 gm

These ingredients are mixed and homogenized in a blender. To this is added the yolk of one egg. A small amount of brandy or rum may be added to give it flavor. Vitamins may be added to the mixture or given parenterally. If the feedings are given chilled, they are easier to swallow. The daily amount supplies approximately 2000 calories.

If the patient can swallow more than just liquids, custard, junket, gelatin creamed soups, puréed vegetables, etc., may be added to the diet.

When the obstruction is complete, a *gastrostomy* must be performed before treatment is started. In borderline cases in which obstruction is imminent and when the patient is unable to swallow a string as a means of aiding the passage of a bougie, a gastrostomy is also indicated. The operation is to be avoided if possible, however, for obvious reasons.

Results of Roentgen Therapy

It is difficult to obtain any clear-cut information about the results of roentgen therapy which can be compared with the large experience with surgical treatment now available. Obviously this is because the choice of patients for the two types of treatment is different. In the majority of instances surgery is given the first chance and roentgen therapy has been confined to those patients whose growths are apparently inoperable or who are too old or who have physical contraindications to surgical interference. No large series of patients treated with roentgen therapy by preference, which might be properly compared with a surgically treated group, is yet available. The most nearly comparable series so far as choice of cases is concerned is that of Buschke and Cantril, but it is small.

From the point of view of survival, the most that can be said is that, whereas formerly without any treatment 100 per cent of the patients died in less than two years after the diagnosis was made, with modern x ray therapy the number who live beyond two years is considerable. Furthermore, there is an increasing proportion of patients, small to be sure, who survive five or more years.

Lefevre reports 10 per cent survival for more than two years without evidence of recurrence or metastasis.

Krebs and associates reported 500 cases of patients treated by the rotational technique. Of these 33.3 per cent died in less than three months, 50 per cent in less than one year, and 90 per cent in less than two years, leaving 10 per cent who survived beyond two years.

Jens Nielson reports improved survival using the rotation technique. It follows: Over one year 25 per cent, as compared with 10 per cent previously, and over two years 15 per cent as compared with 4 per cent previously.

Many authors are able to report a few cases of survival for five or more years with an apparent preponderance in favor of the higher voltage techniques although the total numbers are too small to draw final conclusions. Fleming in a review of the literature collected 800 cases from various sources with ten survivals for five or more years.

So far as palliation is concerned, the results are only fairly satisfactory and so much inferior to those of surgical resection using modern methods as to bear a most unfavorable comparison. The average experience of many workers in this field (Krebs and associates, Moulouquet, Lefevre, Baclesse, Buschke and Cantril, etc.) is the disappearance of the tumor in 20 per cent of the patients treated. This is based upon roentgenological and esophagosopic evidence. It is interesting that in isolated instances in the experience of everyone who uses the method complete disappearance of the tumor has been observed at autopsy. Fried has reported three such cases. The metastases so frequently present however ultimately cause the death of these patients. Many of these 20 per cent of patients develop stenosis as a result of cicatrization and require additional treatment by bougienage.

Deglutition is definitely improved by elimination of the neoplastic stenosis in 4 per cent of the patients reported by Krebs and in a varying number as

Mediastinal and pulmonary fibrosis are very frequent and may even lead to spots in the lung where the blood vessels and lymphatics are completely obliterated by the fibrous contraction. This tendency is also more marked with the multiple field method than with the rotation technique.

Cicatricial contraction of the tumor site as the neoplastic tissue gives way to the ingrowth of fibrous tissue is a frequent occurrence. In fact, with the majority of patients it is necessary to combine bougienage with the roentgen therapy. For this purpose, in all but the relatively unobstructed vegetative type of tumors, it is wise to cause the patient to swallow a thread before the treatment is started in anticipation of subsequent need for dilatation.

Whenever the growth is so advanced that it has actually invaded the wall of the trachea or a bronchus, the end result of treatment as the tumor is destroyed is almost invariably the creation of a fistula. In every patient, therefore, whose tumor is in the juxtatracheal or bronchial segment, a tracheobronchoscopy must be performed, and if the growth is found to be invading the air passage, roentgen treatment should not be given.

Invasion of the wall of the aorta often ends in a fatal hemorrhage as the growth is destroyed by the roentgen rays. For this reason, unusually large tumors in the portion of the esophagus which lies next to the aorta should not be given large doses.

Adjuvant Treatment

Medical treatment for the skin reaction needs no comment.

The maintenance of nutrition during the treatment is of paramount importance. Swallowing is often difficult either because of swelling or because of pain. As mentioned above, both of these discomforts are largely eliminated by the exhibition of Meticorten, 5 mg. every six hours (or four times a day) during the course of treatment. The same precautions must be observed as for any utilization of this steroid hormonal substance.

One must provide a suitable dietary formula which the patient may be able to swallow. That devised by Paterson is as follows:

Milk	1000 cc
Butter	40 gm
Flour	20 gm
Sugar	30 gm

These ingredients are mixed and homogenized in a blender. To this is added the yolk of one egg. A small amount of brandy or rum may be added to give it flavor. Vitamins may be added to the mixture or given parenterally. If the feedings are given chilled, they are easier to swallow. The daily amount supplies approximately 2000 calories.

If the patient can swallow more than just liquids, custard, junket, gelatin creamed soups, puréed vegetables, etc., may be added to the diet.

When the obstruction is complete a gastrostomy must be performed before treatment is started. In borderline cases in which obstruction is imminent and when the patient is unable to swallow a string as a means of aiding the passage of a bougie, a gastrostomy is also indicated. The operation is to be avoided if possible, however, for obvious reasons.

Results of Roentgen Therapy

It is difficult to obtain any clear-cut information about the results of roentgen therapy which can be compared with the large experience with surgical treatment now available. Obviously this is because the choice of patients for the two types of treatment is different. In the majority of instances surgery is given the first chance and roentgen therapy has been confined to those patients whose growths are apparently inoperable or who are too old or who have physical contraindications to surgical interference. No large series of patients treated with roentgen therapy by preference, which might be properly compared with a surgically treated group, is yet available. The most nearly comparable series so far as choice of cases is concerned is that of Buschke and Cantrell, but it is small.

From the point of view of survival, the most that can be said is that, whereas formerly without any treatment 100 per cent of the patients died in less than two years after the diagnosis was made, with modern x ray therapy the number who live beyond two years is considerable. Furthermore, there is an increasing proportion of patients, small to be sure, who survive five or more years.

Lefèvre reports 10 per cent survival for more than two years, without evidence of recurrence or metastasis.

Krebs and associates reported 500 cases of patients treated by the rotational technique. Of these 33.3 per cent died in less than three months, 50 per cent in less than one year, and 90 per cent in less than two years, leaving 10 per cent who survived beyond two years.

Jens Nielson reports improved survival using the rotation technique as follows: Over one year 25 per cent as compared with 10 per cent previously, and over two years 15 per cent, as compared with 4 per cent previously.

Many authors are able to report a few cases of survival for five or more years with an apparent preponderance in favor of the higher voltage techniques, although the total numbers are too small to draw final conclusions. Fleming in a review of the literature collected 800 cases from various sources with ten survivals for five or more years.

So far as palliation is concerned, the results are only fairly satisfactory and so much inferior to those of surgical resection using modern methods as to bear a most unfavorable comparison. The average experience of many workers in this field (Krebs and associates, Moulouquet, Lefèvre, Baclesse, Buschke and Cantrell, etc.) is the disappearance of the tumor in 20 per cent of the patients treated. This is based upon roentgenological and esophagoscopy evidence. It is interesting that in isolated instances in the experience of everyone who uses the method complete disappearance of the tumor has been observed at autopsy. Fried has reported three such cases. The metastases so frequently present, however, ultimately cause the death of these patients. Many of these 20 per cent of patients develop stenosis as a result of cicatrization and require additional treatment by bougienage.

Deglutition is definitely improved by elimination of the neoplastic stenosis in 4 per cent of the patients reported by Krebs and in a varying number as

Mediastinal and pulmonary fibrosis are very frequent and may even lead to spots in the lung where the blood vessels and lymphatics are completely obliterated by the fibrous contraction. This tendency is also more marked with the multiple field method than with the rotation technique.

Cicatricial contraction of the tumor site as the neoplastic tissue gives way to the ingrowth of fibrous tissue is a frequent occurrence. In fact, with the majority of patients it is necessary to combine bougienage with the roentgen therapy. For this purpose, in all but the relatively unobstructed vegetative type of tumors, it is wise to cause the patient to swallow a thread before the treatment is started in anticipation of subsequent need for dilatation.

Whenever the growth is so advanced that it has actually invaded the wall of the trachea or a bronchus, the end result of treatment as the tumor is destroyed is almost invariably the creation of a fistula. In every patient, therefore, whose tumor is in the juxtatracheal or bronchial segment, a tracheobronchoscopy must be performed, and if the growth is found to be invading the air passage, roentgen treatment should not be given.

Invasion of the wall of the aorta often ends in a fatal hemorrhage as the growth is destroyed by the roentgen rays. For this reason, unusually large tumors in the portion of the esophagus which lies next to the aorta should not be given large doses.

Adjuvant Treatment

Medical treatment for the skin reaction needs no comment.

The maintenance of nutrition during the treatment is of paramount importance. Swallowing is often difficult either because of swelling or because of pain. As mentioned above, both of these discomforts are largely eliminated by the exhibition of Meticorten, 5 mg. every six hours (or four times a day) during the course of treatment. The same precautions must be observed as for any utilization of this steroid hormonal substance.

One must provide a suitable dietary formula which the patient may be able to swallow. That devised by Paterson is as follows:

Milk	1000 cc
Butter	40 gm
Flour	20 gm
Sugar	30 gm

These ingredients are mixed and homogenized in a blender. To this is added the yolk of one egg. A small amount of brandy or rum may be added to give it flavor. Vitamins may be added to the mixture or given parenterally. If the feedings are given chilled, they are easier to swallow. The daily amount supplies approximately 2000 calories.

If the patient can swallow more than just liquids, custard, junket, gelatin, creamed soups, puréed vegetables, etc., may be added to the diet.

When the obstruction is complete, a *gastrostomy* must be performed before treatment is started. In borderline cases in which obstruction is imminent and when the patient is unable to swallow a string as a means of aiding the passage of a bougie, a gastrostomy is also indicated. The operation is to be avoided if possible, however, for obvious reasons.

Results of Roentgen Therapy

It is difficult to obtain any clear-cut information about the results of roentgen therapy which can be compared with the large experience with surgical treatment now available. Obviously this is because the choice of patients for the two types of treatment is different. In the majority of instances surgery is given the first chance and roentgen therapy has been confined to those patients whose growths are apparently inoperable or who are too old or who have physical contraindications to surgical interference. No large series of patients treated with roentgen therapy by preference, which might be properly compared with a surgically treated group, is yet available. The most nearly comparable series so far as choice of cases is concerned is that of Buschke and Cantrell, but it is small.

From the point of view of survival, the most that can be said is that, whereas formerly without any treatment 100 per cent of the patients died in less than two years after the diagnosis was made, with modern x-ray therapy the number who live beyond two years is considerable. Furthermore, there is an increasing proportion of patients, small to be sure, who survive five or more years.

Lefèvre reports 10 per cent survival for more than two years, without evidence of recurrence or metastasis.

Krebs and associates reported 500 cases of patients treated by the rotational technique. Of these 33.3 per cent died in less than three months, 50 per cent in less than one year, and 90 per cent in less than two years, leaving 10 per cent who survived beyond two years.

Jens Nielson reports improved survival using the rotation technique as follows: Over one year 25 per cent, as compared with 10 per cent previously, and over two years 15 per cent, as compared with 4 per cent previously.

Many authors are able to report a few cases of survival for five or more years with an apparent preponderance in favor of the higher voltage techniques, although the total numbers are too small to draw final conclusions. Fleming in a review of the literature collected 800 cases from various sources with ten survivals for five or more years.

So far as palliation is concerned, the results are only fairly satisfactory and so much inferior to those of surgical resection using modern methods as to bear a most unfavorable comparison. The average experience of many workers in this field (Krebs and associates, Moulouquet, Lefèvre, Baclesse, Buschke and Cantrell, etc.) is the disappearance of the tumor in 20 per cent of the patients treated. This is based upon roentgenological and esophagoscopy evidence. It is interesting that in isolated instances in the experience of everyone who uses the method complete disappearance of the tumor has been observed at autopsy. Fried has reported three such cases. The metastases so frequently present, however, ultimately cause the death of these patients. Many of these 20 per cent of patients develop stenosis as a result of cicatrization and require additional treatment by bougienage.

Deglutition is definitely improved by elimination of the neoplastic stenosis in 4 per cent of the patients reported by Krebs and in a varying number as

reported by others Moulouguet and Mallet report that the majority of their patients are relieved

The former also reports suppression of pain in 68 per cent and improvement in general condition with gain in weight in 74 per cent of his patients

Treatment by Cobalt Bomb Irradiation

Another source of external irradiation which has come into limited use in recent years is the so-called cobalt 'bomb' unit or Theratron This apparatus produces gamma rays which are focused upon the tumor at approximately 80 cm distance It has an advantage similar to that of the rotating chair technique for roentgen therapy in that the 'bomb' or radiation source can be made to rotate a full circle around the patient By proper centering of the surface upon which the patient lies, the center of the arc of the rotation can be made to coincide with the exact position of the patient's lesion

Not enough experience with the use of this form of treatment is yet available to make comparisons with other sources of irradiation so far as the results are concerned One advantage is that the original cost of installation is considerably less than that of the supervoltage roentgen machines

Conclusion

From what has been said, it is obvious that irradiation therapy of carcinoma of the esophagus has made progress, while radium therapy has fallen into disfavor and deep roentgen therapy has become the method of choice The reasons for this have been discussed Improvements in depth dosage using higher voltage apparatus and greater precision of application including rotational techniques now make it possible to obtain a two-year survival in at least 10 per cent of the patients Furthermore, even if a cure cannot be foreseen, an impressive palliative effect and in some instances the eradication of the stenosing lesion may be obtained Deglutition is made possible and the need for a gastrostomy is postponed or eliminated, all of which has a considerable influence upon the morale of the patient

Further progress will certainly be made in this field, but it will always be subject to the need for an earlier diagnosis else the early appearance of metastases and the rapid invasion of vital organs and tissues must always render the prognosis grave in this highly malignant tumor

To date it must be admitted that, although it carries with it a greater mortality than roentgen treatment, surgical extirpation as currently practiced offers better prospects of long survival and more effective palliation

APPENDIX

Diets for Patients with Various Degrees of Obstructive Dysphagia

The regimens described below were devised to meet the needs of patients with swallowing difficulties. They are derived from the *Diet Manual of the Massachusetts General Hospital*. Ordinarily liquid or soft diets are ordered with the result that, as swallowing becomes more difficult, the patient drifts into an inadequate diet, or eats improper foods which will lodge in the throat causing irritation and swelling that increases the obstruction.

Esophageal Regimen 1 provides an adequate diet for the patient who can tolerate only very thin liquids. The liquids in this diet have been strained through a wire mesh sieve having about 25 mesh to the inch and should be easily swallowed. An abundant supply of vegetables and raw fruits prepared so that they may be easily taken is essential.

Esophageal Regimen 2 is planned for the patient who can swallow some soft foods as well as liquids, therefore any of the liquids listed in Regimen 1 plus the soft feedings (strained through a No. 25 sieve) listed in Regimen 2 may be used.

Important Instructions for Both Regimen 1 and 2

1. Take only the foods listed in the diet. Use no breadstuffs, cake or crackers.
2. Strain all foods through a 25 mesh wire sieve. * Custards, junkets, gelatin and ice cream which are absolutely smooth foods need not be strained on Regimen 2.
3. Hold all foods, even liquids, in the mouth until they are well mixed with the saliva.
4. Let frozen desserts, such as ice cream, melt in the mouth before swallowing them.
5. Alternate milk with other-than milk feedings as much as possible in order to avoid curds forming, thereby increasing the obstruction.
 - Take 6 ounces of orange juice or tomato juice and 2 ounces of meat juice or liver pulp daily. Meat juice or liver pulp may be served cold in tomato juice or in warm bouillon. If the meat juice or liver pulp is overheated, coagulated particles which form from overheating will be difficult to swallow.
7. Make feedings as palatable as possible. Use salt liberally. The recipes under suggested feedings should be varied to suit the needs and the tastes of

* The Waring Blender manufactured by Waring Products Corporation, 23 West 43rd Street, New York 36, New York, has been found to be an excellent device to liquefy foods so that they will readily pass through a No. 25 sieve.

the patient The caloric content of the diet is determined by the patient's tolerance and tastes

8 In order to increase the vitamin and iron content of the diets, fortified cereals such as Ralston's Super Farina, Gerber's cereal, or 5 minute Cream of Wheat may be used in preparing the feedings The protein content of both Regimen 1 and 2 may be increased by the addition of powdered milk to the foods

ESOPHAGEAL FEEDINGS—REGIMEN 1

(Thin liquids strained through No. 25 sieve)

Suggested feedings to be taken one feeding an hour when patient is awake

- Cereal water (1 cup milk—1 tablespoon flour barley rice wheat etc.—) or 2 tablespoons sugar—2 ounces light cream)
- Strained fruit juices (6 oz. fruit juice plus sugar or karo and lemon juice to taste)
- Tomato and vegetable juices from cooked vegetables or extracted vegetable juices
- Beef juice or liver pulp (2 oz. of either in 4 oz. bouillon or in 4 oz. tomato juice flavored with lemon and steak sauce)
- Bouillons—clear soups—chicken broth
- Tea—coffee—soft drinks—milk
- Milk shakes (3 oz. cream—3 oz. milk—1 or 2 tablespoons sugar—may be flavored with vanilla coffee, or banana or ice cream added)
- Malted milks (6 oz. milk—2 tablespoons malted milk—may be flavored with vanilla coffee or cocoa—1 or 2 tablespoons sugar)
- Chocolate milk (3 oz. cream—3 oz. milk—1 tablespoon chocolate syrup)
- Eggnog (3 oz. cream—3 oz. milk or 6 oz. milk—1 egg—1 to 2 tablespoons sugar—may be flavored with vanilla, coffee, cocoa etc.)
- Ovaltine
- Cocoa (4 oz. cream—4 oz. milk—1½ tablespoons sugar—1 tablespoon cocoa)
- Cream soups (½ cup light cream—½ cup puréed vegetable or juice and seasonings—butter may be added)
- Oyster stew strained (3 oz. milk—3 oz. cream—4 or 5 oysters which are strained out—seasonings)
- Strained chowders
- Lemonade (Lemon juice and heavy sugar syrup with water)

SUGGESTED MEAL PLAN FOR REGIMEN 1

Approximate Calories—3100 (C 280 gm, P 75 gm, F 190 gm)

7 A.M.	6 oz. sweetened citrus juice with lemon juice
8 A.M.	10 oz. cereal water
9 A.M.	8 oz. broth
10 A.M.	6 oz. malted milk
11 A.M.	6 oz. tomato juice
12 noon	8 oz. cream soup
1 P.M.	6 oz. fruit juice
2 P.M.	6 oz. milk shake
3 P.M.	8 oz. bouillon plus beef juice or liver pulp
4 P.M.	6 oz. eggnog
5 P.M.	8 oz. oyster stew (strained) or cream soup
6 P.M.	6 oz. citrus juice (sweetened lemon juice)
7 P.M.	8 oz. cream soup
8 P.M.	6 oz. cocoa

ESOPHAGEAL FEEDINGS—REGIMEN 2

Any of the liquids listed in Regimen 1 plus soft feedings such as the following strained through a No. 25 sieve

Creamed minced chicken or meat	Strained vegetables
Creamed minced fish	Strained fruits
Mashed potato	Plain cornstarch puddings
Raw liver pulp in tomato juice	Junkets
Plain Jello or bavarian cream	Custards
	Plain ice cream or sherbets

N B Use no breadstuffs, cake, cookies or crackers

SUGGESTED MEAL PLAN FOR REGIMEN 2

Approximate Calories—3000 (C-300 gm P 100 gm F 150 gm)

7 A M	Strained cereal with milk or cream Cocoa
8 A M	Strained orange juice with sugar and lemon juice
10 A M	Cereal water or lemonade
12 noon	Creamed minced chicken Mashed potato with butter and cream Strained spinach with butter Milk with part cream Soft custard pudding
2 P M	Chocolate malted milk
4 P M	Tomato juice—6 oz plus beef juice or liver pulp—2 oz
5 P M	Welsh rarebit (no bread or crackers) Strained beans Milk with cream Strained peaches
8 P M	Eggnog with cream

Tube Feedings for Patients with a Gastrostomy or a Jejunostomy

The accompanying table (Fig. 409) represents a pattern for a basic tube feeding which can be modified to meet the needs of the individual patient.

The ingredients should be mixed in the order listed in a Waring Blender, adding at first only enough of the cream to facilitate this process. The remaining cream should then be added and the mixture blended only until smooth. Finally, the mixture should be strained.

In order to prevent clogging of the tube, the feeding may be diluted with 2 parts of water and mixed thoroughly.

Modifications of Basic Tube Feedings

In order to meet the needs of the individual patient, the Basic Tube Feeding or the Low Sodium Basic Tube Feeding recipe may be modified easily as shown below.

- (1) *High Caloric Modification*
Sugar or oil may be added or the feeding may be diluted with dextrose and water rather than plain water. The volume of feeding to be given to the patient may be increased.
- (2) *Low Caloric Modification*
The amount of sugar, oil or cream used is decreased or the volume of feeding given is diminished.

Food	Total	Grams			Cal	Grams			Mg			I U		Milligram			
		C	P	I		Ca	Phos	Fe	Na	K	Vit A	Asc Acid	Thi amin	Ribo-flavin	Nia cin		
Basic Tube Feeding																	
Cottage cheese	100 gm	2.0	19.5	0.5	90.5	0.096	0.189	0.3	290	72	20	—	0.020	0.310	0.1		
Eggs—coddled	two	0.6	12.2	11.0	150.2	0.052	0.202	2.6	81	100	1,100	—	0.080	0.260	trace		
Strained baby meat 3-3½ oz cans	10½ oz	—	54.8	10.7	315.5	0.034	0.472	13.2	693	192.1	—	—	0.031	0.693	10.4		
Vit Pent	1 cc	—	—	—	—	—	—	—	—	—	8,333	83	1.600	1.600	16.0		
Sugar	200 gm	199.0	—	—	796.0	—	—	—	—	—	—	—	—	—	—		
Cream 20	240 cc	9.6	7.2	48.0	499.2	0.232	0.184	trace	72	218.4	1,992	trace	0.072	0.336	trace		
Total		211.2	93.7	70.2	1851.4	0.414	1.047	16.1	1136	582.5	11,445	83	1.803	3.199	26.5		
800 cc. Basic Tube Feeding		210	95	70	1850												
Low Sodium Basic Tube Feeding																	
Cottage cheese salt free	100 gm	2.0	19.5	0.5	90.5	0.096	0.189	0.3	20	39.0	20	—	0.020	0.310	0.1		
Eggs—coddled salt free	two	0.6	12.2	11.0	150.2	0.052	0.202	2.6	81	100.0	1,100	—	0.080	0.260	trace		
Strained baby beef salt free 3-3½ oz cans	10½ oz	—	54.8	10.7	315.5	0.034	0.472	13.2	78	296.1	—	—	0.031	0.693	10.4		
Vit Pent	1 cc	—	—	—	—	—	—	—	—	—	8,333	83	1.600	1.600	16.0		
Sugar	200 gm	199.0	—	—	796.0	—	—	—	—	—	—	—	—	—	—		
Cream 20	240 cc	9.6	7.2	48.0	499.2	0.232	0.184	trace	72	218.4	1,992	trace	0.072	0.336	trace		
Total		211.2	93.7	70.2	1851.4	0.414	1.047	16.1	251	653.5	11,445	83	1.803	3.199	26.5		
800 cc. Low Sodium Basic Tube Feeding		210	95	70	1850												

FIGURE 409 Basic Tube Feeding Patterns

- (3) *High Protein Modification*
The amount of meat ■ increased
- (4) *Low Protein Modification*
The content of meat cottage cheese or egg is decreased
- (5) *Higher Calcium Modification*
Milk powder ■ substituted for cottage cheese and milk is added

Storage and Use of All Tube Feedings

Cases of diarrhea have been traced to the use of tube feedings which were not properly refrigerated. *It is imperative that all tube feedings be kept under refrigeration at all times and not be left outside the refrigerator between feedings.*

Each time before it is used, the bottle of feeding should be inverted and well shaken.

It is advisable to store half a recipe of tube feeding in a quart bottle to prevent unnecessary exposure to room temperature and to facilitate the shaking process.

BIBLIOGRAPHY

Note The following bibliography is arranged by chapter and occasionally by subdivision of material within a chapter. The documentation for each chapter begins with the original citation of the second edition (1951) of *Les Maladies de L'Oesophage*. These are then followed by other and more recent references. Both the original French references and the newer references are arranged chronologically.

Chapter 1 Anatomy

- Rouvière (H.) *Anatomie des lymphatiques de l'homme* Masson et Cie édit. Paris 1932
- Coulouma et van Varseveld. L'innervation vago-sympathique de l'oesophage. *S I C I C* Lille 1938
- Bradley Small Wilson et Walten. Considérations anatomiques à propos de la section des nerfs pneumogastriques. *J A M A* 133 17 15 fév 1947
- Pesce (G.) Ricerche istologica sui rapporti esofago-diaframmatica nei feti umani. *Boll d malat d orecchio* no 7-8 luglio agosto 1948 ■ 182
- Calvet (J.) et Poulhès (J.) Les artères de l'oesophage. *Ann d oto laryngol* t 65 no 7 1948
- Rubaltelli (E.) Distribution des nerfs laryngés. Contribution histo anatomofonctionnelle a la connaissance de l'innervation de l'oesophage de la trachée et de l'espace trachéo laryngo-oesophagien chez l'homme. *Arch ital tracheobroncho-esofagoscopia* no 3 1939
- Guibert (H. L.) et Fabre (L.) L'advenue de l'oesophage terminal. *L O R L internat* juin 1944
- Guernier (Y.) et Marchal (G.) A propos de l'innervation du segment inférieur de l'oesophage. *L O R L internat* juin 1944
- Fabre (L.) Les nerfs extrinsèques de l'oesophage terminal. *L O R L internat* juin 1944
- Guibert (H. L.) et Fabre (L.) Le système nerveux intrinsèque de l'oesophage terminal. *L O R L internat* juin 1944
- Walters (Waltman) Neibling (H.) Bradley (W.) Small (J.) et Wilson (J. W.) Distribution of the vagus nerves at lower end of the oesophagus. *Quart Rev of O R L and Broncho Esophagology* 7 1 mars 1948
- Stohr (J.) Observation sur les muscles et le tissu de l'oesophage. *Klin Wochenschr* Berlin 1948 26 7-8 p 128
- Davis (E. D. D.) Anatomie appliquée et physiologie du pharynx et de l'oesophage. *Ann Royal College of Surgeons of England* London 1948 3/3 pp 139-153
- Cortesi. Réflexions sur la structure de la paroi postérieure du pharynx et du debut de l'oesophage. *L O R L italiana* 1948 fasc IV
- Liefooghe (J.) Quelques aspects anatomiques des pneumogastriques à la partie inférieure de l'oesophage. *Société méd et anatomo clin de Lille* 30 juin 1948. In *J des Sc med de Lille* no 14 3 juil 1949
- Jackson (R. G.) Anatomie des nerfs pneumogastriques dans la région inférieure de l'oesophage et dans la région gastrique. *Anatomical Record* Philadelphie 103 1 janv 1949 pp 1-18
- Van Geertruyden (J.) Anatomie chirurgicale du plexus vagal péri oesophagien. *Arch d Anat d Histol et d Embryol* Strasbourg 32 fasc 4-8 pp 231-238
- Resano (J. H.) Anatomie chirurgicale de l'oesophage thoracique. Les deux triangles du médiastin postérieur. *Presse méd* 57 2 5 janv 1949
- Swenson (O.) Merrill (K. Jr) Peirce (C.) et Rhemlander (H. F.) Apport sanguin et nerveux de l'oesophage. *J Thoracic Surg* 19 3 mars 1950
- Guernier (Y.) et Marchal (G.) Les chaînes sympathiques collatérales. *C R Ass anatomistes* Louvain 1950

- Rosenberg (D) Estudo anatómico dos nervos vagos na porção infrabronquica do esôfago (Anatomical study of the 10th pair of the cranial nerves in the infrabronchial portion of the esophagus) *Arch chirug clin exp* 13(3) 189-259 1949
- Shapiro (A L.) and Robillard (G L.) The esophageal arteries. Their configurational anatomy and variations in relation to surgery. *Ann Surg*, Philadelphia 1950 131/2 and *Ar de cirugía*, Buenos Aires 12(2) 171, 185, 1950
- Swigart Lav (I) Siekert (R G) Hambley (W C) and Anson (R J) The esophageal arteries. An anatomic study of 150 specimens *Surg, Gynec & Obst*, 90(2) 234-243 1950 (234-243)
- Potter (S E) and Holyoke (E A) Observations on the intrinsic blood supply of the esophagus *Arch Surg* 61(5) 944-948, 1950
- Perrotin (J) and Faurel (J) Les artères de l'œsophage thoraco-abdominal (The arteries of the thoracoabdominal esophagus) *Rev chir (Paris)* 70(3-4) 93-104 1951
- Conni (G) and Passarelli (L) Contributo alla conoscenza dell'esofago umano I La vascolarizzazione dell'esofago dal punto di vista anatomico funzionale (A study of the human esophagus I The vascularization of the esophagus from anatomical and functional points of view) *Arch chir (Torace)* 5(8) 269-286 1951
- Bertocchi (A) Omegna (G) and Negretti (S) Studio anatomico chirurgico sulle arterie dell'esofago (Study on the surgical anatomy of the arteries of the esophagus) *Chirur gia* 6(2b) 177-224, 1951
- Bracci (U) Principe (U) and Belluci (G) La vascolarizzazione dell'esofago (The arterial vascularization of the esophagus) *Arch ital ant embriol* 56(1) 21-38 1951
- Fumagalli (P) Costituzione dei nervi vaghi periesofagei (Constitution of the periesophageal vagus nerves) *Minerva chir (Torino)* 7(11) 429-437 1952

Chapter 2 Physiology

- Bossi et Piero La température normale de l'œsophage chez l'homme *Bronchoscopie œsophagoscopie et gastroscopie*, Paris 4, 3 juil 1937
- Huet et Nemours Auguste Etude radio physiologique du premier temps de la déglutition isolée *Presse méd* 1943 p 125
- Cherubino Sur les mouvements de contraction des parois œsophagiennes par suite des excitations de distension à l'exclusion du réflexe pharyngé de la déglutition *Il a'la'* 17, 5 mai 1945
- Nemec (V) Eructation œsophagienne aéro-œsophagie *Casopis lékařu českých* Prague 86 11 1947, pp 405-406
- Brunetti Recherches kymographiques sur la déglutition *Rec de laryngol*, janv fév 1948
- Davis (E D D) Anatomie appliquée et physiologie du pharynx et de l'œsophage *Ann Royal College of Surgeons of England Londres* 3 sept 1948 p 139
- Kramer (I) et Ingelfinger (F S) Motilité de l'œsophage humain chez des témoins et chez des malades souffrant de troubles œsophagiens *Am J Med* New York 7 2 août 1949 pp 168-173
- Williams (A F) La sensibilité de l'œsophage après sympathectomie *Thorax* 5 1 mars 1950
- Cannon (W B) Acid closure of cardia *Am J Physiol* 23 105-114 1908
- Robins (S A) and Jankelson (I R) Cardio esophageal relaxation *J A M A* 87 1961-1964 1926
- Cannon (W B) Law of denervation *Am J Med Sc* 198 737-750 1939
- Nemours Auguste (S) L'extrémité inférieure de l'œsophage normal (The inferior part of the normal esophagus) *Presse méd* 57 65 1949
- Lerche (W) *The Esophagus and Pharynx in Action A study of structure in relation to function* 229 pp Springfield Illinois Thomas 1950
- Kramer (P) and Ingelfinger (F J) Cardiospasm generalized disorder of esophageal motility *Am J Med* 7 174-179 1949
- Idem Esophageal sensitivity to Mecholyl in cardiospasm *Gastroenterology* 19 242-253 1951
- Bariatti (R) and Taccani (C) Ricerche di fisiopatologia cardiaca (il tono del cardia) (Physiopathological studies of the cardia [The cardiac tonus]) *Arch chir (Torace)* 6 (1) 1-66 1952
- Palmer (E D) Attempt to localize normal esophagogastric junction *Radiology* 60 875-881 1953
- Hillemand (P) Beau (H) and Bernard (H) Etude critique de certains aspects de l'extrémité inférieure de l'œsophage (A critical study of certain aspects of the lower end of the esophagus) *Presse méd* 61(61) 1229-1231 1953
- Sanchez (G C) Kramer (P) and Ingelfinger (F J) Motor mechanisms of esophagus particularly of its distal portion *Gastroenterology* 25 321-332 1953

- Colombo (C) Beatrice (E) and Bianchi (M) Rilevi manometrici nel segmento diaframmatico cardiale dell'esofago (Findings at manometry in the phrenocardial segment of the esophagus) *Atineta chir* (Torino) 1954
- Ingelfinger (F J) Kramer (P) and Sanchez (G C) Gastroesophageal vestibule its normal function and its role in cardiospasm and gastroesophageal reflux *Am J Med Sc* 78 417-425, 1954
- Ingelfinger (F J) Benign disorders of the lower esophagus *Bull New England Med Center* 16(1) 8-12 1954
- Marchand (P) Gastro esophageal sphincter and mechanism of regurgitation *Brit J Surg* 47 504-513 1955
- Poppel (M H) Lentuno (W) Zaima (C) and Jacobson (H) Closing mechanism of lower esophagus in man *JAMA* 161(3) 196-198 1956
- Hightower (N C) Newer concepts of the physiology of deglutition and dysphagia *Am J Surg* 93 2 154-162 1957
- Wilkins (E W Jr) Current considerations of esophageal physiology normal and abnormal *Net England J Med* 257(1) 24-31 1957

Chapter 3 Roentgen Examination of the Normal Esophagus

- Cannon Des premières applications des rayons X dans l'étude du tube digestif *JAMA* 67 1 3 janv 1914
- Ramond Borrien et Jacquelin Sur l'abouchement de l'œsophage dans l'estomac *Bull et mém de la Soc anat de Paris* 1921 p 204
- Palugyay Etudes radioscopiques sur la déglutition œsophagienne *Arch f die ges Phys CC* 5-6 620 1923
- Caballero et Nemours Auguste Mécanisme de la déglutition *Bull et mém de la Soc franç d électroth et de radiol* 1924 p 33
- Belot et Lepennetier La radiographie en série de l'œsophage *Bull de la Soc de rad méd de France* mars 1925 no 7 p 72
- Parkinson et Bedford (D) L'empreinte de l'artère pulmonaire sur l'œsophage *The Lancet* 15 août 1931 p 337
- Palugyay Röntgenuntersuchung und Strahlenbehandlung der Speiseröhre Springer Vienne 1931
- Israelski (M) et Simchowitz (H) Sur le diagnostic radiologique du relief muqueux de l'œsophage *J de radiol* mai 1932 p 196
- Béclère L'examen radiologique en position pieds au plafond *Soc Rad méd de France* 10 mai 1932 — Perrin Grangerard et Salesse *Paris méd* 12 août 1933
- Bétoulières (P) Contribution radiologique à l'étude de l'œsophage normal *Thèse Montpellier* 1934
- Lelong et Aimé (P) *Soc de Péd* 21 nov 1933 *Soc de gastro entérol* 8 janv 1934
- Pizon Etude radiologique de l'œsophage de l'adulte *Thèse Paris* 1936
- Cendreau et Jutras (A) Exploration radiologique de l'œsophage de l'estomac et du petit intestin avec substances opaques en sac de caoutchouc *Union méd du Canada* 67 119 1938
- Huet (P C) et Nemours Auguste Etude radio physiologique du premier temps de la déglutition isolé *Arch de mal de l'app dig* 32 5-6 mai juin 1943
- Bakwin (H) Galenson (E) et Le Vine (B E) Roentgenographic appearance of the esophagus in normal infants *Am J Dis Child* 68 oct 1944 pp 243-247
- Mathey Cornat (R) Sur l'exploration radiologique de la partie basse de l'hypopharynx et de la bouche de l'œsophage (Anatomie et Physiologie radiologiques) *J de radiol* 1947 p 428
- Latraverse (V) Un procédé aidant l'examen radiologique du segment gastro œsophagien *Am J Dig Dis* 14 9 1947 pp 298-302
- Templeton (F E) Movements of the esophagus in the presence of cardio spasm and other esophageal diseases A roentgenologic study of muscular action *Gastro Enterology* 10 96-101 1948
- Nemours Auguste L'extrémité inférieure de l'œsophage normal *Presse méd* 65 21 oct 1949 pp 960-962
- Iselin (H) Scopie œsophagienne *Schweiz med Woch* Bale 79 48 3 déc 1949 pp 1146-1149
- Jutras (A) Levrier (P) et Longtin (M) Etude radiologique de l'œsophage paradiaphragmatique et du cardia *J de radiol* 30 7-8 1949 pp 373-416

- Templeton (F E) *X Ray Examination of the Stomach. A description of the roentgenologic anatomy, physiology and pathology of the esophagus stomach and duodenum* 520 pp Chicago, Illinois University of Chicago Press 1944
- Mauri (C) and Catalano (D) Sull'impiego di un colinergico nella esplorazione radiologica del tratto inferiore dell'esofago (The use of a cholinergic agent in the radiological investigation of the lower part of the esophagus) *G Ital chir* 9(8) 527-532 1953
- Bourdon (R) Massare (C) and Thubeau (A) A propos du reflux gastro-œsophagien, un sign nouveau la double colonne (The double column. A new radiological sign of gastro-œsophageal reflux) *Gaz méd Fran e* 61(22) 1335-1340 1954
- Kraus (R) and Strand (I) Hat die Oesophagus-kymographie eine präoperative Bedeutung für den Thoraxchirurgen? (Has esophagus kymography a preoperative value in thoracic surgery?) *Thoraxchirurgie* (Stuttgart) 3(4) 319-333 1955
- Nadal (R) Debray (C) and Jourde (L) Nouvelle méthode d'examen radiologique de l'œsophage Utilisation d'un ganglioplegique (méthanthéline) (A new method of radiological examination of the esophagus Use of a ganglioplegic (methanthel line)) *Arch mal Appar dig* 44(5) 633-638, 1955
- Poppel (M H), Lentino (W), Zaino (C) and Jacobson (H) Closing mechanism of lower esophagus in man radiological study of 500 unselected patients *JAMA* 161 196-198 1956

Chapter 4 Esophagoscopy and Esophagoscopic Biopsy

- Tucker (G) The retrograde esophagoscopy *Surg Clin N Amer* fév 1924
- Moulouguet (A) Un nouvel œsophagoscope *Ann d'oto laryngol*, 1940 p 71
- Scalori (C) Considérations sur la technique de l'œsophagoscopie *Rev de laryngol* déc. 1941
- Torre (P) L'œsophagoscopie rétrograde Etude d'un nouveau procédé Thèse Paris 1942
- Aubin (A) Un nouveau procédé d'œsophagoscopie rétrograde *Ann d'oto laryngol*, 1943 p 90
- Moulouguet (M A) Un nouvel œsophagoscope *Soc de Laryngol des Hôp de Paris* 15 mars 1943 in *Ann d'oto laryngol* no 4-5-6 avril mai juin 1944 p 71
- Ray Heford (A) Un œsophagoscope amélioré *Rev Gastro Enterol*, Boston 16 11 nov 1944
- Henry (M) Un nouvel œsophagoscope *Proc Royal Soc of Med* Londres 39 12, oct 1946
- Soulas (A) Technique de la bronchoscopie et de l'œsophagoscopie Présentation d'instrument *Congrès de la Soc. franç d'O R L* oct 1946
- Holinger (P) Nouvel œsophagoscope et bronchoscope pour l'enfant *L O R L internat*, déc 1947 p 191
- Resano (J H) L'œsophagoscopie dans les affections de l'œsophage *El Dia medico*, Buenos Aires 1947 pp 237-248
- Robb (J M) Une modification de l'œsophagoscope de Negus *The Laryngoscope* Saint Louis 1947, 57/3 pp 243-246
- Boros (Edwin) Œsophagoscope au moyen d'un instrument flexible Un nouvel œsophagogastroscope *Gastro-Enterol*, Baltimore, 1947, 8/6, pp 724-728 2 fig *Gastro-Enterol* 1948, 11/6 pp 879-882
- Guillermín (Fouzard et Drouard) Considérations sur l'œsophagoscopie rétrograde A propos d'un cas infranchissable par le haut *Soc d'O R L de Lyon et de la Région* 23 mai 1948
- Martin (G E) La position de la broncho-œsophagologie en Grande Bretagne *Arch Otolaryngol*, Chicago, 47 6, juin 1948, pp 705-720
- Schandler (R) Le diagnostic de sûreté par œsophagoscopie optique *JAMA*, 138 12, 20 nov 1948
- Soulas (A) Appareil cinématographique endo-œsophagien *Bull Soc de broncho-œsophagol et de gastrol de Langue franç* an 1948
- Holinger (P H), Anson (G C) et Johnston (K C) Cinématographie bronchoscopique et œsophagoscopique *J Thoracic Surg* 1948 17/2 pp 179-188
- Holinger (P H) Progress in esophagology *Arch Otolaryng* Chicago 47, 2 fév 1948 pp 119-126
- Butt (W A) et Boyes (H W) L'anesthésie pour la bronchoscopie et l'œsophagoscopie *Canadian Med Asso J* Montréal 60 1 janv 1949, pp 62-64
- Hufford (A Ran) Optique flexi rigide pour l'œsophage *Gastro-Enterol*, mars 1949
- Holinger (P H) La photographie endoscopique en broncho-œsophagologie II *Congrès d'Oto-Laryngol* Londres, juil 1949
- Aubin (A) L'œsophagoscopie rétrograde *Congrès franç d'oto-rhino-laryngol* 20 oct 1949
- Iselin (H) L'œsophagoscopie *Schweiz med Wochenschr* Bâle, 79 48 3 déc 1949

- Jannouli (G. E.) Ein neues Ösophago Tracheoskop aus plastischer Masse *Practica O R L* 12, fasc 3 1950 p 185
- Barlow (D.) Distally illuminated œsophagoscopy telescope *Brit Med J* London 4651 1950 487
- Grump et Henning Le diagnostic du cancer de l'œsophage La technique de la biopsie par curettage rétrograde *Gastro Entérol* Baltimore 1949 13 2 pp 153-159
- Bocca (E.) L'anestesia endotracheale in Chirurgia otorinolaringoiatrica *Arch ital di otolo* Milano 61 maggio 1950 supp III
- Villata (I.) Modifications électro-cardiographiques au cours de l'œsophagoscopie pratiquée sous anesthésie avec anesthésie locale et générale *Arch ital di otolo* Milano 61 maggio 1950 supp III
- Andersen (H. A.) McDonald (J. R.) et Olsen (A. M.) Le diagnostic cytologique du cancer de l'œsophage et du cardia *Sem d hôp de Paris* 28 fév 1950 p 739
- Terraol (J.) et Guibert (H. L.) La biopsie de l'œsophage par écouvillonnage *Presse méd* no 21 5 avril 1950 pp 371-372
- Savonuzzi (G.) and Ferranti (L.) Nuovo metodo per esaminare estemporaneamente la mucosa esofagea (A new method for quick examination of the esophageal mucosa) *Atti del II Congresso Na- di chir torac* Roma 7 148-151 1951
- Smith (C. C. K.) and Tanner (N. C.) The complications of gastroscopy and œsophagoscopy *Brit J Surg* 43(180) 396-463 1956
- Som (M. L.) and Arnold (L. M.) Esophagoscopy in the diagnosis and treatment of esophageal diseases *Am J Surg* 93(2) 183-204 1957

Chapter 6 Congenital Malformations

CONGENITAL ATRESIA AND TRACHEO ESOPHAGEAL FISTULA

- Gamble (H. A.) Tracheo-esophageal fistula Description of a new operation and case report *Ann Surg* 107 mai 1938 pp 701-707
- Imperatori (C. J.) Fistule œsophago-trachéale congénitale sans atrésie de l'œsophage *Archiv Oto-Laryngol* sept 1939
- Shaw Correction chirurgicale de l'atrésie congénitale de l'œsophage avec fistule trachéo œsophagienne *J Thoracic Surg* 9 2 déc 1939
- Lanman (T. H.) Congenital atresia of the esophagus A study of thirty two cases *Arch Surg* 41 nov 1940 pp 1060-1083
- Ashley (J. D.) Jr Congenital atresia of the esophagus with tracheo esophageal fistula Case report *Radiology* 36 mai 1941 pp 621-624
- Siegel Atrésie congénitale de l'œsophage *Zentralbl für Gynakol* Leipzig an 65 22 31 mai 1941
- Leven (N. L.) Congenital atresia of the esophagus with tracheo esophageal fistula Report of successful extrapleural ligation of fistulous communication and cervical esophagostomy *J Thoracic Surg* 10 aout 1941 pp 648-657
- Ozzano (T.) Contributo alla conoscenza dell'atrésie congenita dell'esofago *Minerva Med* sept 1941
- Carter (B. N.) Operation for the cure of congenital atresia of the esophagus *Surg Gynec & Obst* 73 oct 1941 pp 485-491
- Selander (E.) Röntgenbefunde bei congenitaler Ösophagus Atresie *Acta radiol* 27 1941 pp 802-807
- Fuhrman (M.) Dragutsky (D.) Rook (G.) et Grossman (L.) Congenital atresia of the esophagus *Radiology* 38 mars 1942 pp 326-329
- Hall (G.) Congenital atresia of the esophagus *Med J Australia* 1 11 avril 1942 p 440
- Jorge (G.) et Latenda (R.) Deux cas de malformation congenitale de l'œsophage *Arch Societ argent de anatomia normal y patol* Buenos Aires 4 1 2 3 4 5 mai sept 1942
- Fuhrman (M.) Dragutsky (D.) Rook (G.) et Grossman (L.) Aeroesophagophotography in congenital atresia of the esophagus *Radiology* 39 juil 1942 pp 100-102
- Chont (L. K.) et Starry (L. J.) Congenital atresia of the esophagus Report of four cases *Radiology* 40 fév 1943 pp 169-174
- Haight (C.) et Towsley (H. A.) Congenital atresia of the esophagus with tracheo esophageal fistula Extrapleural ligation of fistula and end to end anastomosis of esophageal segments *Surg Gynec & Obst* 76 juin 1943 pp 672-688
- Poth (F.) Atrésie congénitale de l'œsophage *Surgery* Saint Louis 8 6 déc 1943
- Singleton (A. O.) et Knight (M. D.) Congenital atresia of the esophagus with tracheo-esophageal fistula Transpleural operative approach *Ann Surg* 119 avril 1944 pp 556-572

- Humphreys (G) Le traitement chirurgical de l'atrésie congénitale de l'œsophage *Surgery* 8, 5, mai 1944
- Haight (C) Congenital atresia of the esophagus with tracheo-esophageal fistula Reconstruction of esophageal continuity by primary anastomosis *Ann Surg*, 120, oct 1944 pp 623-651
- Daniel (R. A. Jr) Congenital esophagus, with tracheoesophageal fistula *Am Surg*, 120 nov 1944 pp 764-771
- Ladd Traitement de l'atrésie congénitale de l'œsophage *Ned. England J Med*, 230, 25 mai 1944 pp 625-637
- I even (N. L.) et Lamm (B. G.) Congenital atresia and congenital tracheo-esophageal fistula *Lancet*, 65, mai 1945 pp 179-181
- Holt (J. F.), Haight (C.) et Hodges (F. J.) Atrésie congénitale de l'œsophage et fistule trachéo-bronchique *Radiology*, 47 5, nov 1946
- Mannon (R. P. O.) Atrésie partielle congénitale de l'œsophage associée à un diverticule congénital *Radiology*, 47, 5, nov 1946 pp 470-477
- Ten Cate (J.) Atrésie congénitale de l'œsophage *Nederl tijdschr voor geneesk*, Amsterdam 90, 50, 14 déc 1946
- Morel et Jacquemet Malformation œsophago trachéale chez un nouveau né *Soc de chir de Lyon* 19 déc 1946
- Kjelland (P. M.) Atrésie œsophagienne congénitale avec fistule œsophago-trachéale *Acta paediatrica* 33 2 1946 pp 151-157
- Gross (R. E.) et Scott (H. W.) Correction of esophageal atresia and tracheo-esophageal fistula by closure of fistula and oblique anastomosis of esophageal segments *Surg Gynec & Obst*, 82 5 mai 1946
- Lam (C. R.) Le traitement chirurgical de l'atrésie congénitale de l'œsophage avec fistule œsophago-trachéale *Surgery* 20, 2 1946 pp 174-179
- Sobel (A.) et Sobel (P.) A propos d'un cas d'imperforation congénitale de l'œsophage *J de radiol et d'électrol* Paris 27 9, 1946 p 428
- White (M. I.) et Birdsong (Mc L.) Atrésie congénitale de l'œsophage avec fistule trachéo-œsophagienne Anastomose bout à bout réussie *Surgery* 20 4 1946 pp 548-557
- Luste et Petit Un nouveau cas d'atrésie œsophagienne *Acta paediatrica Upsala* 1 3-4, 1946-1947 pp 184-186
- Tavernier Atrésie congénitale de l'œsophage chez le nouveau né *Soc de chir de Lyon* 17 déc 1946 in *Lyon chir* 42 2 mars avril 1947
- Ladd (W. F.) et Swenson (O.) (Boston) Atrésie œsophagienne et fistule trachéo-œsophagienne *Ann Surg*, 125 1, janv 1947 pp 23-40
- Lam (C. R.) Nouveau cas de traitement chirurgical du rétrécissement congénital de l'œsophage avec fistule trachéo-œsophagienne *Quart Rev O R L and Broncho Esophagol* mars 1947
- Dauplex Atrésie congénitale de l'œsophage *Société d'O R L de Paris* 21 avril 1947, in *Presse méd* 29 nov 1947, no 61
- Haight (C.) Atrésie congénitale de l'œsophage avec fistule œsophago-trachéale *Surg Gynec & Obst* 84 4 avril 1947 pp 504-506
- Davis (M.) Atrésie congénitale de l'œsophage *Canadian Med Assoc J* Montréal 56 5 mai 1947
- Holt (John) Haight (C.) et Hodges (F.) Sténose congénitale de l'œsophage avec fistule trachéo-œsophagienne *Quart Rev. of O R L*, juin 1947
- Franklin (R.) Atrésie congénitale de l'œsophage *Lancet* Londres 257 6468 16 août 1947
- Swenson (O.) Anastomose termino terminale de l'œsophage pour atrésie œsophagienne *Surgery*, 22 2, août 1947
- Longmire (W.) Jr Atrésie congénitale et fistule trachéo-œsophagienne Quatre cas consécutifs d'anastomose œsophagienne primitive réussie *Arch of Surg*, Chicago 55 3 sept 1947
- Saunders (C. H.) et Wright (H.) Atrésie congénitale de l'œsophage *Canadian Med Assoc J* Montréal 57, 3 sept 1947
- Perrone (J. A.) Fleming (W. H.) et Whitaker (T. R.) Atrésie congénitale de l'œsophage avec fistule trachéo-œsophagienne *Arch Oto-Laryng* 46 5 nov 1947
- Truchet L'imperforation congénitale de l'œsophage *Lyon chir* 47 6 nov-déc 1947
- Groult et Loy Absence partielle de l'œsophage *L O R L internat* déc 1947 p 147
- Lelong Le diagnostic précoce de l'atrésie œsophagienne *Arch franç de pédiat* Paris 4 5 1947
- Moniserrat (J.) Malformation congénitale de l'œsophage Fistule trachéale *Arch 5^e argent de Ana normal y path*, Buenos Aires 1 6 sept in *J de ch* = 63 7-8 1947
- Nicod (M.) Vomissements des nourissons dus à une malformation œsophagienne *Ann Pédiat Basel* 169 1947 pp 313-314

- Bloch (A) et Autier (Ch) Malformation œsophagienne congénitale chez un mongolien
Ann d O L, 1947, p 502
- Debré (R) Leveuf (Jacques) Tieffry (St) et Mallet (R) L atrésie œsophagienne avec
abouchement de l'œsophage anormal dans la trachée *Soc de pédiat* III janv 1948 *Sem*
d hôp no 13 26 fév 1948
- Eerland (L) Atrésie de l'œsophage avec fistule œsophago trachéale *Neder tijdschr voor*
geneesk no III 10 janv 1948
- Bocca et Bergier Vomissements chez un nourrisson atteint de rétrécissement congénital de
l'œsophage *Soc de pédi de la Région provençale* 11 janv 1948 in *Sem des hôp* no 30
III avril 1948
- Mallet, L atrésie œsophagienne *Diagnostic médical d'urgence J des praticiens* 61 5 29
janv 1948
- Langman (J) Atrésie de l'œsophage avec fistule œsophago trachéale *Nederlan tijdschr voor*
geneesk Haarlem 9^e 5 29 janv 1948
- Baumann Traitement chirurgical de l'atrésie congénitale de l'œsophage avec fistule œso
trachéale *Presse méd* 7 fév 1948
- Franklin Œsophage et atrésie congénitale *Ann Royal College of Surgeons of England*
Londres 2 2 fév 1948 pp 57-110
- Méras (H) et Coignat (J) L atrésie congénitale de l'œsophage avec fistule trachéo-œsopha
gienne *Ga des hôp* no 10 6 mars 1948
- Guilleminet Geay Jacquet et Batmaz (Simon) Atrésie congénitale de l'œsophage avec
fistule œso trachéale *Soc de chir de Lyon* 8 avril 1948 in *Lyon chir* 1948
- Sandblom (P) Traitement chirurgical de l'atrésie œsophagienne *Nordisk med* Stockholm
38 16 16 avril 1948 pp 787-791
- Adam (H) Les indications thérapeutiques dans les malformations congénitales reconnues a
la naissance *Concours méd* 23 avril 1948 p 927
- Lyon (L G) et Johnson (S G) Atrésie congénitale de l'œsophage et fistule trachéo-œsoph
agienne *J Thoracic Surg*, 17 2 avril 1948 pp 162-177
- Szenes (H) Kongenitale Œsophagusstenose *Mischr Ohrenh* 82 4 avril 1948 pp 168-171
- Rienhoff (W F) Transplantation pré thoracique de l'estomac dans le traitement des atrésies
congénitales de l'œsophage thoracique *Bull Johns Hopkins Hosp* III 4 avril 1948
pp 496-499
- Waegle (R) et Poitevin (J P) A propos d'un cas d'atrésie congénitale de l'œsophage chez
un nouveau né *Soc de gyn et d'obst* Strasbourg 5 juin 1948
- Ingelrens Venlerenberghe et Lacheret Atrésie de l'œsophage avec fistule œso-trachéale
Soc de chir de Lille 9 juil 1948
- Lisa (J) et Taylor (H) Malformation congénitale de l'œsophage sans fistule trachéo-
œsophagienne *Net York State J Med* 48 14 15 juil 1948
- Sandblom (P) Le traitement de l'atrésie congénitale de l'œsophage d'un point de vue
technique *Acta chir Scand* Stockholm 97 4 30 sept 1948
- Bartolomucci (E) L'atrésie de l'œsophage Diagnostic d'urgence et possibilité de traitement
chirurgical *La Diagnosi* Naples 4 9 sept 1948 pp 347-350
- Draps (H) Un cas de malformation de l'œsophage *Bruxelles méd* Bruxelles 28 nov 1948
pp 2499-2503
- Byron (F) Atrésie congénitale de l'œsophage avec hypoplasie ou agénésie du segment in
férieur *Surgery* 24 5 nov 1948
- Schreiber (G) Une malformation du nouveau né curable chirurgicalement mais souvent
méconnue L'atrésie œsophagienne *Concours méd* 28 nov 1948 p 387
- Fontan Verger Dabadie Moules et Martin Atrésie congénitale de l'œsophage avec fistule
œsophago-trachéale *J méd de Bordeaux et du Sud Ouest* 125 11 nov 1948
- Waegle (R) et Poitevin (J P) Deux cas d'atrésie congénitale de l'œsophage chez le nouveau
né *Strasbourg méd* 108 36 25 déc 1948
- Crislan (J) Atrésie œsophagienne avec fistule œsophago-trachéale chez le nouveau né
Arch franç de pédi Paris 5 1 1948
- Despons Bentegeat et Dabadie Malformation congénitale trachéo-œsophagienne *J méd*
Bordeaux 175 1948 p 322
- Meloche (L) Anesthésie pour un cas III atrésie congénitale de l'œsophage avec fistule trachéo-
œsophagienne *Bull de l'Ass d méd de Langue franç du Canada* Montréal 77 1 1948
pp 86-87
- Fribert (O) L'anesthésie dans les opérations pour atrésie œsophagienne *Nordisk med* 38
16 1948 pp 791-792
- Kluska (Vladimir) Kongenitalni atresia œsophagu *Pediatricke Listy* no 2 1948

- Stursova Cudova (J) Dva případy kongenitální atresie oesophagu *Pediatricke Listy* no 2 1948
- Sweet (R H) Nouvelle méthode du rétablissement de la continuité du canal alimentaire dans les cas d'atrésie congénitale de l'oesophage avec fistule trachéo-oesophagienne non traitée par anastomose primitive immédiate *Ann Surg*, 127, 4, 1948 pp 757-768
- Lambert (M D) Le diagnostic de l'atrésie congénitale de l'oesophage dès les premières heures de la vie *Thèse*, Paris, 1948
- Fèvre (Marcel) Malformations congénitales de l'oesophage In *Encyclop méd chir Pédiatre* 1948 vol 1, p 4017
- Franklin (R H) 3 enfants opérés avec succès pour atrésie congénitale de l'oesophage avec fistule oesophago trachéale *Proc Royal Society of Medicine* 11 fév 1949
- Chigot A propos d'un cas d'atrésie de l'oesophage chez un nouveau né *Soc nat franç de gastro entérologie*, 14 fév 1949
- Petit et Lepage Atrésie de l'oesophage chez un nouveau né Intervention Guérison *Soc de péd* 26 avril 1949
- Fèvre (Marcel) Atrésie oesophagienne du nouveau né *Acad de chir*, 27 avril 1949
- Barcat Atrésies oesophagiennes *Acad de chir*, 27 avril 1949
- Chigot et Esteve Atrésie de l'oesophage chez le nouveau né *Acad de chir* 27 avril 1949
- Perruchio Atrésie oesophagienne avec fistule oesotrachéale Intervention *Mort Acad chir*, 27 avril 1949
- Perruchio, Barcat, Chigot, Laurence (G) Duhamel (B) et Brochantz Sur l'atrésie oesophagienne du nouveau né *Acad de chir* 27 avril 1949
- Laurence, Duhamel et Brochantz Atrésies oesophagiennes *Acad de chir* 27 avril 1949
- Jemerin (E E) et Halkin (C R) Atrésie congénitale de l'oesophage *Ann Surg* 129 4 avril 1949
- Handousa Bey (A S) Some rare anomalies of the oesophagus with discussion of their aetiology *J Lar Otol*, Londres, 63 4 avril 1949, pp 217-224
- Hussenstein (J) Atrésie congénitale de l'oesophage avec fistule trachéo-oesophagienne *Ann Surg* 129 5 mai 1949, pp 572-587
- Bigger (I A) Le traitement de l'atrésie congénitale de l'oesophage avec fistule trachéo-oesophagienne *Ann Surg* 129 5 mai 1949, pp 572-587
- Frank (R) et Paul (L) Un cas de dédoublement congénital de l'oesophage *Radiology*, 53, 3 sept 1949, pp 313-468
- Chigot (P L) L'atrésie congénitale de l'oesophage *Le Phare méd de Paris* mai juin 1949
- Chigot (P L) Essai de traitement d'une atrésie congénitale de l'oesophage chez un nouveau né *Arch mal app dig*, 38, 5-6, mai juin 1949
- Ashe (W M) et Seybold (W D), Huit cas d'atrésie congénitale de l'oesophage avec fistule oesotrachéale traités par anastomose primitive bout à bout des deux segments de l'oesophage *Proc Staff Meet Mayo Clin* 24 12 8 juin 1949, pp 327-333
- Ingalls (T H) et Prindle (R A) Atrésie oesophagienne avec fistule trachéo-oesophagienne causes épidémiologiques et tératologiques *New England J Med* 240, 25, 23, juin 1949 pp 987-995
- Coe (H E) Oesophageal atresia and tracheo-oesophageal fistula *Bull Vancouver M Ass* 25 9 juin 1949, pp 191-193
- Lam (C R) Atrésie de l'oesophage traitée par des interventions multi-étapées *J Thoracic Surg* 18 3 juin 1949 pp 327-336
- Lemoine et Ribera Conduite à tenir en présence de certaines malformations de l'oesophage chez le nouveau né *Soc de lar d hôp de Paris* 11 juil 1949 In *Presse méd* 26 nov 1949
- Geruz (T Cl) Atrésie oesophagienne Reconstruction de la continuité de l'oesophage intra thoracique par l'opération trans pleurale (en deux fois) *Acta paediatrica Upsala* 37 5-6 1949
- Vernejoul (R de) L'atrésie congénitale de l'oesophage et son traitement chirurgical *Concours méd* 6 août 1949
- Sacrez (R) Forster (G) et Wiener (S) Deux cas de malformation oesophagienne avec fistule trachéo-oesophagienne *Soc de péd de l'Est* 19 nov 1949
- Lemoine (J) et Ribera Conduite à tenir en présence de certaines malformations congénitales de l'oesophage chez le nouveau né *Ann d'oto laryngol*, 66 12 déc 1949
- Chigot Atrésie de l'oesophage *J de chir*, 65 10-11 1949
- Chigot (P L) et Esteve Atrésie de l'oesophage avec fistule trachéo-oesophagienne *Arch franç de péd* Paris 6, 2 1949
- Ricard (P M) et Vezina (N) Atrésie congénitale de l'oesophage avec fistule trachéo-oesophagienne, ligature extra pleurale de la fistule et anastomose bout à bout des segments oesophagiens *Union méd Canada* 78 7, 1949 p 804

- Huizinga (E.) Œsophagus Atresie mit œsophagotrachealer Fistel *Ned tydschr geneesk* 1949 295
- Laugmann (J.) Atresie œsophagea mit œsophagotrachealer Fistel *Ned tydschr geneesk* 1949 pp 373-378
- Ingelrans (P.) Vanlerenberghe et Lacheretz (M.) Atrésie congénitale de l'œsophage avec fistule œso-trachéale du segment inférieur *Lille chir* janv fév 1950
- Fontan Verger Dabadie Bouet et Couteau Atrésie œsophagienne du nouveau né *Soc de Méd de Bordeaux* 12 oct 1949 in *Arch franç de pédi* 7 3 1950 p 333
- Cadenaule Gautret Dabadie et Marchand Atrésie œsophagienne avec large communication œsophago-trachéale *Ibid* p 334
- Fontan Laumonier Verger et Marchand Atrésie œsophagienne du nouveau né *Ibid* p 334
- Roberts (F. N.) La cure de l'atrésie congénitale de l'œsophage *South African Med J* Cape town 24 10 11 mars 1950
- Kahle (H. R.) Fistule trachéo-œsophagienne Un cas traité avec succès et ligature bout à bout *Rev. Orleans Med and Surg J*, 10^e 10 avril 1950 pp 487-492
- Morelle L'atrésie congénitale de l'œsophage *Soc de chir de Lyon* 25 mai 1950

- Langman (J.) L'atrésie de l'œsophage avec une fistule œsophago-trachéale (Atresia of the esophagus combined with esophagotracheal fistula) *Acta neer morphol norm & Pathol* Utrecht 6(3) 287 1948
- Ingalls (T. H.) and Prindle (R. A.) Esophageal atresia with tracheo-esophageal fistula Epidemiologic and teratologic implications *New England J Med* 240(25) 987-995 1949
- Langman (J.) Esophageal atresia and esophageo-tracheal fistula *Acta neerl Morphol norm & pathol* 6(4) 308-314 1949
- Ladd (W. E.) Congenital anomalies of the esophagus *Pediatrics* 6(1) 9-19 1950
- Butler (C. L.) and Ende (M.) Double esophagus with carcinoma in one Report of a case with autopsy *Arch Path* 49(5) 605-611 1950
- Potts (W. J.) Esophageal atresia and tracheo esophageal fistula *Med Clin North America* Jan 1950 pp 247-256
- Van de Walle (J.) L'atrésie de l'œsophage III L'anesthésie du nouveau né (Esophageal atresia III Anesthesia in the newborn) *Acta pædiat belg* 5(1) 46-49 1951
- Longmire (W. P. Jr.) Antethoracic jejunal transplantation for congenital esophageal atresia with hypoplasia of the lower esophageal segment *Surg Gynec & Obst* 93(3) 310-316 1951
- Potts (W. J.) Atresia of the esophagus with or without tracheo-esophageal fistula *Postgrad Med* 10(4) 304-309 1951
- Shaw (R. R.) Paulson (D. L.) and Siebel (E. A.) Congenital atresia of the esophagus with tracheo-esophageal fistula Treatment of surgical complications *Ann Surg* 142(2), 204-213 1955

CONGENITAL STENOSIS AND ESOPHAGEAL WEBS

- Chavanne (L.) Double sténose congénitale de la partie moyenne de l'œsophage *Ann d oto laryngol* no 1-2 1940
- Paissieu Rouget et Nguyen Khac Vien Rétrécissement congénital de l'œsophage *Bull Soc méd d hop de Paris* 16 mai 1941
- Aubin et Clerc Rétrécissement congénital de l'œsophage *Ann d O R L* 12 oct déc 1945
- Mauerhofer (H.) et Riniker (P.) Contribution à la connaissance de la sténose œsophagienne congénitale *Practica O R L* Bale 7 1 1945 pp 33-82
- Pinsolle Contribution à l'étude du rétrécissement congénital de l'œsophage *Thèse Bordeaux* 1945
- Despons Le rétrécissement congénital de l'œsophage *Rev de laryngol* fév mars 1946
- Bannon (R. P. O.) Sténose partielle congénitale de l'œsophage associée à un diverticule congénital *Quart Rev. O R L* juin 1946
- Aubertin Lartigaut et Tessier Anomalies du tube digestif chez le nourrisson *J de méd de Bordeaux et du Sud Ouest* 123 17-18 sept 1946
- Cannizzaro (C.) Su di caso di stenosi esofagea congenita *Atti XXI Congrès Soc ital O R L* Venezia 1946
- Guisez (J.) Quelques formes de sténoses graves non cancéreuses de l'œsophage *Congrès de la Soc franç d O R L* oct 1946
- White (A.) et Birdsong (Mc L.) Rétrécissement congénital de l'œsophage avec fistule trachéo-œsophagienne *Surgery* 20 4 oct 1946 pp 548-557
- Holt Haight et Hodges Sténose œsophagienne congénitale avec fistule trachéo-œsophagienne *Radiology* nov 1946 p 457

- Guilleminet Oblitération congénitale de l'œsophage par diaphragme membraneux *Soc de chir de Lyon* 12 déc 1946 *m J de chir* 64, 1-2 1948
- Wildenberg (Van den) Malformations congénitales de l'œsophage *Bull de l Acad de m'd de Belgique* 11, 7 1946 pp 203-212
- Aubin (A) et Clerc (C) Quelques cas difficiles de rétrécissement de l'œsophage *Ann O R L*, 13 1946 p 448
- Mauerhofer (Herbert) Diagnostic différentiel entre sténose congénitale de l'œsophage et hernie de l'hiatus œsophagien *Practica O R L* 8, 5-6, 1946
- Bloch (A) et Autier (C) Malformation œsophagienne congénitale chez un mongolien *Sy d O R L de Paris* 21 avril 1947, in *Presse méd* no 69 29 nov 1947
- Colebatch Sténose congénitale de l'œsophage *Med J Australia* Sydney 34 19 10 mai 1947
- Franklin Sténose congénitale de l'œsophage *Lancet*, 16 août 1947, pp 243-244
- Russell Verbrycke (J) Jr Traitement du rétrécissement œsophagien *Am Practitioner* 2-4 1947 pp 263-268
- Lam (C R) Nouveau cas de traitement chirurgical du rétrécissement congénital de l'œsophage avec fistule trachéo œsophagienne *Ann d oto laryngol* 1947 p 614
- Flynn (R) Rétrécissement de l'œsophage *Ann d oto laryngol* 1947, p 616
- Autier (Ch) 68 cas de rétrécissements de l'œsophage *Ann d oto laryngol* 1947 p 587
- Aberg (S) Cas de sténose familiale de l'œsophage *Ann d oto laryngol* 1947, p 228
- Guilleminet et Lacour Atresies et sténoses œsophagiennes congénitales par diaphragme membraneux *Pédiatrie* 36 5-6 1947, pp 197-205
- Bocca Carrega et Berger (P) Vomissements chez un nourrisson atteint de rétrécissement congénital de l'œsophage *Soc de pédi de la Région provençale* 11 janv 1948
- Szenes (H) Kongenitale Oesophagusstenose *Monats fur Ohrenh* 82 4 avril 1948 pp 168-171
- Sandblom (P) La réparation plastique de la sténose congénitale de l'œsophage *Acta chir Scand* Stockholm 97, fasc 4 30 sept 1948
- Szenes (X H) Sténose œsophagienne inhabituelle *Monats fur Ohrenh* Vienne 82 3 1948 pp 138-139
- Wildenberg (Van den) Contribution à l'étude des rétrécissements congénitaux de l'œsophage et des diverticules de l'œsophage *Ann Oto Laryngol* 65 7, 1948 pp 390-397
- Champeau Henrot et Tomatis Sténose congénitale du tiers inférieur de l'œsophage opérée par voie transpleurale à l'âge de neuf mois *Acad de chir* 18 mai 1949 *Ann Oto Laryngol* 66 10-11 oct nov 1949
- Guillermmin Touzard et Protar Considérations sur l'œsophagoscopie rétrograde à propos d'un cas de rétrécissement infranchissable par le haut *Lyon m'd*, 187 27, 3 juil 1949 p 13
- Filip (D) Notes sur quatre cas de sténose congénitale de l'œsophage II *Congrès intern d oto laryngol* Londres 18-23 juil 1949
- Holinger (P H) Johnston (K C) et Grengard (J) Anomalies congénitales de l'œsophage révélées par des corps étrangers œsophagiens *Am J Dis of Child* 78 4 oct 1949 pp 467-476
- Mounier Kuhn Charleux (G) et Bretton Sur les dysphagies de l'enfant les sténoses congénitales de l'œsophage à symptomatologie tardive (3 observations) *Soc de broncho-œsophagol et de gastroc de Langue franç*, 14 mai 1950
- Morel Jacquemet and Guilleminet Oblitérations congénitales de l'œsophage par diaphragme membraneux (Congenital esophageal atresia due to membranous diaphragm) Une observation de malformation œsophagotrachéale chez un nouveau né (A case of esophago tracheal malformation in a newborn) *Lyon chir*, 47(2) 210-212 and 213-214 1947
- Bugden (W F) and Delmonico (J E Jr) Lower esophageal web *J Thoracic Surg* 31(1) 1-18 1956
- Vargas (L L) Britton (R C) and Goodman (E N) Congenital esophageal stenosis Report of a case of annular muscle hypertrophy at the esophagogastric junction *Net England J Med* 255 26 1224-1227 1956
- Bugden (W F) Lower esophageal web *Am J Surg* 93(2) 248-251 1957

BRACHY ESOPHAGUS (CONGENITALLY SHORT ESOPHAGUS WITH THORACIC STOMACH)

- Findlay (L) et Brown Kelly Congenital shortening of the œsophagus and the thoracic stomach resulting therefrom *Royal Soc of Med (Section Laryngology)* 5 juin 1931 et *J Laryngol and Otol* 46 12 déc 1931 pp 797-816

- Akerlund *Acta radiol* 6 mars 1932
- Aguna (H) Valero (M) et Filippi (T F de) Ulcera de œsophage Un syndrome piolroco y anemia hypoplasico en une nino de dos annos *Arch argent de Pédi* juil 1932
- Clerf (H L) et Manges (W F) Congenital anomalies of the esophagus with special reference to the congenitally short esophagus with a portion of stomach above the diaphragm *Ann O R L* 47 1933 p 1058
- Monkhouse (J P) et Montgomery (S K) A report of seven cases of partial thoracic stomach with short esophagus *J Laryng* 48 1933 p 743
- Wright (A J) Congenital shortening of esophagus *Proc Roy Acad of Med* 26 1933 p 1325
- Truesdale (P E) Diaphragmatic hernia at the esophageal hiatus the short esophagus and thoracic stomach *New England J Med* 212 1935 p 240
- Clerf (H L) et Manges (W F) Brévité congénitale de l'œsophage *Rev of Gastro-Enterol* 1935 p 1
- Hatt Raccourcissement congénital de l'œsophage *Arch Oto-Laryng* nov 1936
- Blasingame (F J L) Œsophage congénitalement raccourci (estomac thoracique) *Ann Surg* 103 fasc. 4 no 3 mars 1936 pp 337-347
- Gillespie (J B) Raccourcissement congénital de l'œsophage *J Pediat* 8 1936 p 38
- Vervloet Brévité congénitale de l'œsophage *Ned tijdschr voor geneesk* 1936
- Gerlings (P G) Congenital deformities of the esophagus *Acta Pediat* 19 3 12 avril 1937 pp 370-391
- Ross (J P) The congenital short esophagus *St Bartholomews Hosp J* 41 1939 p 223
- Lelong Aimé et Bernard (J) Le brachy-œsophage avec estomac partiellement thoracique *Bull Soc méd d hôp de Paris* no 4 3 fév 1939 p 195 *Arch franç de pédi* 1943 p 152
- Froment (J) Mayson Bourret et Vallier Ectopie de tout l'estomac dans l'hémithorax droit avec œsophage court et hernie colique *Lyon méd* 163 24 avril 1939 *Presse méd* 22 fév 1939
- Briggs (P) Dick (J) et Herst Ulcère simple et brachy-œsophage *Proc Roy Soc Med* 32 sept. 1939
- Myles (R. B) Brachy-œsophage familial *Brit J Radiol* 12 nov 1939
- Cajano (A) Etude des malformations congénitales de l'œsophage *Acta radiol* mai 1940
- Ribadeau Dumas (L) Chambrun (J) et Walter (R) Brachy et méga-œsophage avec estomac entièrement thoracique *Soc de pédi* Paris 19 nov 1940
- Schmut Sur un cas de brachy-œsophage du nourrisson *Thèse Paris* 1940
- Lelong et Aimé (P) Contribution à l'étude des vomissements du nourrisson 6 cas de brachy œsophage *Presse méd* 1940
- Lesné Ronget (Mille) et Longeaux Un cas de brachy-œsophage avec œsophagite ulcéreuse *Bull Soc méd d hôp de Paris* no 41-44 6 janv 1941 p 687
- Lelong Lamy et Aimé Un nouveau cas de brachy-œsophage chez le nourrisson *Presse méd* 28 janv 1941 p 106
- Rossier Un cas de brachy-œsophage chez un nourrisson *Soc de pédi* Paris 20 mai 1941 in *Bulletin* 38 5 mai 1941 pp 184-187
- Polley (H F) 47 cas de brachy-œsophage avec estomac thoracique et hernie de l'hiatus *JAMA* 116 1941 pp 821-825
- Nadaud Brachy-œsophage latent chez un enfant *J de radiol et d'electrol* 24 1942
- Mayoux (R) Chavanne et Eyssautier Un cas de brachy-œsophage *LO R L internat* no 2 fév 1943
- Lelong (M) Brachy-œsophage et hernie diaphragmatique de l'estomac sont deux affections différentes *La Médecine* mars 1943 p 7
- Julien Marie Mallet et Piot Brachy-œsophage et dolicho-colon *Arch franç de pédi* 1 3 1943 p 137
- Calvet (J) et Pallot Sur les diverses malformations des dérivés pharyngiens (trachée bronches œsophage thymus) dans l'imperforation de l'œsophage documents histologiques In *interprétation embryologique LO R L internat* avril 1944
- Nada Kovacic Un cas de brachy-œsophage dans la maladie de Marfan *Ann de pédi de Bale* 165 1 juil 1945 p 32
- Bardier Gouzy et Lacapère Un cas de brachy œsophage *Soc de med de Toulouse* janv 1946
- Land (P T) Congenital short esophagus with thoracic stomach *J Laryng* Londres 61 12 dec. 1946
- Howe (T O) Short esophagus with thoracic stomach *J Laryng* Londres 61 12 dec 1946
- Bertoye (P) Bernheim et Moron (M) A propos du brachy-œsophage chez le nourrisson. *Arch franç de pédi* 3 5 1946 p 485

- Köhler (C) et Papillon Un cas de brachy-œsophage avec hernie rubrösitaire trans-diaphragmatique chez un enfant de 12 jours *Arch franç de pédi*, 3, 5 1946 p 485
- Moron Contribution à l'étude du brachy-œsophage chez le nourrisson *Thèse Lyon* 1946
- Wyllie (W G) et Field (C F) *Arch Dis Child*, 121 108 déc 1946 pp 218-224
- Sée (G) Malformation diaphragmatique ou brachy œsophage observée au cours de quatre générations successives *Soc de pédi*, Paris 17 juin 1947
- Lefebvre (Gérard) Hématémèses récurrentes et périodiques chez un nourrisson porteur d'un brachy-œsophage *So de Gastro Entérol de la Région du Nord* 22 juin 1947, in *Arch mal app dig* 36, 1947
- Soulas (A) Brachy œsophage congénital avec sténose et estomac sus diaphragmatique *Bull et mem Soc méd d hop de Paris* 9 juil 1947
- Lelong (M) Le brachy-œsophage chez le nourrisson *Congrès de pédi*, New York, 14-17 juil 1947
- Lelong (Marcel) Le brachy-œsophage chez le nourrisson *Pediatrics Danubiana* 11 2 nov 1947
- Resano L œsophage court congénital dans la chirurgie *Acad de chir* 15 oct 1947
- Lelong (M) Le brachy-œsophage chez le nourrisson Conférence à la Clinique pédiatrique de l'Hôpital Stefania (Lisbonne) 20 mai 1947 *Rev portuguesa de pediatri e ginecologia* (sous presse)
- Wamberg (E) Sur le brachy œsophage *Acta Paediatrica*, 34 fasc 4 20 nov 1947, pp 293-305
- Bertuglia (B) Sur un cas de brièveté de l'œsophage *Ann di radiol diagnost*, Bologne, 19 6 1947
- Rodriguez et Ramirez Deux cas d'œsophage court avec estomac thoracique partiel *Rev méd de Chili* Santiago, 74 6 juin in *J de chir*, 63, 9 1947
- Castele (van de) Hernie du hiatus diaphragmatique brachy œsophage *Ned tydschr voor geneesk*, Haarlem 90, 25 22 juin in *J de chir* 63, 3, 1947
- Hennebert (P E) Raccourcissement congénital de l'œsophage avec hernie gastrique (à propos de deux cas) *Soc belge d'O R L* 1er fév 1948
- Fugazzola (F) Brachy-œsophage congénital *Minerva medica* Turin, 1, 10 10 mars 1948 pp 260-261
- Olsen (A) et Harrington (S) Hernies de l'hiatus œsophagien du type œsophage court considérations étiologiques et thérapeutiques *J Thoracic Surg* 17, 2 avril 1948
- Chapital (J) Brunel (D) Pierni et Cabanettes (Mme) Brachy œsophage et gastrite ulcéreuse Etude diagnostique *Soc d'Sci med de Montpellier*, 21 mai 1948, in *Montpellier méd* 1948
- Dickson (W) Brièveté congénitale de l'œsophage *Proc Roy Soc of Med* Londres 41 6 juin 1948
- Putney (F J) Estomac thoracique dû à une hernie de l'orifice œsophagien et à un brachy œsophage congénital *Ann Intern Méd* Lancaster, 28 6 juin 1948 pp 1094-1105
- Hennebert (P) Un cas de raccourcissement congénital de l'œsophage *Acta Otorhinolaryngol Belgica*, Bruxelles 2 3 sept 1948, pp 273-277
- Keiser (D) L'œsophage court *Nederl Tydschr voor geneesk* Haarlem 92 51, 18 déc 1948
- Rennu (J B) Short œsophagus *Gastroenterologia* Basel 73, 4-5, 1948, pp 294-298
- Umdensiock (M R) Sur un cas de brachy-œsophage *Arch d mal de l'app dig* 37 1948 p 796
- Hauser (F) Brachy œsophage *Ann paediatr* Basel 170 1948 pp 330-331
- Resano (J H) et Stricht (J van den) L'œsophage court *Bruxelles méd* 29 16 17 avril 1949
- Terracol (J) Les sténoses de l'œsophage thoracique Rapport au Congrès internat Londres juil 1949
- Verhaeghe (A), Lormiaux (A), Belbenoit (Cl) et Dumez (L) Image anormale de la base droite par hernie diaphragmatique avec estomac thoracique et brachy-œsophage *Soc de méd du Nord* séance du 30 sept 1949
- Rennu (J B), Land (F T) et Park (S D S) The short œsophagus a review of 31 cases *Brit Med J* 2 4642, 24 déc
- Stephens (H B) The problem of the acquired short œsophagus report of 18 patients *California Med* 71, 6 déc 1949
- Gutteridge (E) Cardiospasme avec estomac thoracique et brièveté congénitale de l'œsophage *Arch int Oto-Laryng* 51 1 jan 1950
- Lafargue (P) et Doutre (L P) Hernie de l'hiatus œsophagien et brachy-œsophage *J de méd de Bordeaux et du Sud Ouest* 127 1 jan 1950
- Resano (J H) Etude clinique et chirurgicale du brachy-œsophage (à propos de 50 cas per

- sonnels d'œsophages courts) (Clinical and surgical study of short œsophagus [with reference to 50 personal cases]), *Sem hôp. Paris* 76(21) 931-944, 1950
- De Maëstri (A) and Corradi (G) Importanza della diagnosi precoce e della terapia nel brachiesofago (The importance of early diagnosis and treatment in short œsophagus) *Minerva Pediatrica* Turin 2(1) 41-45 1950
- Santy (P) Michaud (P) and Margotton (R) A propos du brachy œsophage (Brachy œsophagus) *Lyon chir* 47(6) 667-680 1952
- Pettersson (G) Hiatal hernia brachy œsophagus and incompetence of the cardia in children *Acta chir scand* 107(5) 321-326 1952
- Chapter 7 : Alterations in Position, Changes in Contour, and Compression of the Esophagus**
- Bonomini (G) Sulla disfagia lusoria *Atti Soc med chir Padova* 213 1945 p 187
- Mayoux (R) et Martin (H) Adénopathie médiastinale de primo infection compression œsophagienne *Lyon méd* 78 1 6 janv 1946
- Gross (R E) Traitement chirurgical de la dysphagia lusoria *Ann Surg* 124 3 sept 1946 pp 532-534
- Tucker (G) Congenital cyst of the mediastinum producing compression of the lower trachea and œsophagus *Ann Otol Rhin and Laryng* 55 3 1946 pp 693-694
- Sweet (R) Findlay (Ch Jr) et Rejersbach (G) Le diagnostic et le traitement de l'obstruction de la trachée et de l'œsophage par un anneau vasculaire congénital *J Pediatrics* 30 1 janv 1947
- Edwards (J) Segment rétro-œsophagien de l'arc aortique gauche ligament artériel droit et aorte descendante droite formant un anneau vasculaire congénital autour de la trachée et de l'œsophage *Proc Staff Meet Mayo Clin* 22 5 3 mars 1947
- Paris (J) Scoliose importante de l'œsophage par lésion pulmonaire apicale droite sans scoliose trachéale *Soc méd du Nord* 7 juin 1947 in *Semaine d'hôp* Paris no 34 14 sept 1947
- Strutz (L) Dysphagia lusoria *Klin Wochenschr* 1947 pp 846-848
- Halpert (B) Snoddy (W) Rohan (K) et Freede (Ch) Arc aortique droit avec anneau vasculaire constricteur de l'œsophage et de la trachée *Arch Pathol* 45 5 mai 1948
- Isaacson (M) Anéurysme du myocarde cause d'obstruction œsophagienne *New Orleans Med & Surg J* 100 12 juin 1948
- Jachimowicz (S) et Szamborski (J) Un cas rare de grosse tumeur carcinomateuse de la tête du pancréas sans ictere ni ascite avec métastases dans les ganglions bronchiques comprimant l'œsophage *Polski tygodnik lekarski* Varsovie 4 9 28 fév 1949
- Herlyn (K E) Dysphagia lusoria bei doppeltem Aortenbogen (Dysphagia lusoria with re duplication of the aortic arch) *Beitr klin Chir* 180(3) 459-464 1950
- Exalto (J) Dicke (W K) and Aalsmeer (W C) De clinische verschijnselen veroorzaakt door de dubbele aortaboog (Clinical signs caused by double aortic arch) *Ned tijdschr voor geneesk* 3(39) 2798-2803 1950
- Nubar (J F) Double aortic arch *J Thoracic Surg* 22(2) 208-215 1951
- Eckstrom G and Sandblom (P) Double aortic arch *Acta chir Scand* 102(3) 183-202 1951
- Issel (W) Speiseröhrenverlauf bei Wirbelsäulenverkrümmungen diagnostische und therapeutische Schwierigkeiten bei dadurch bedingten Stenosen (Diagnostic and therapeutic difficulties in esophageal stenoses due to spinal deformities) *Arztl Wschr* 6(3) 56-59 1951
- Dodrill (F D) Double aortic arch *Surgery* 31(2) 204-211 1952
- Facquet (J) Israel (R) Alhomme (P) and Combaz Deux cas de dysphagia lusoria par crosse aortique double incomplète Guérison par le traitement chirurgical (Dysphagia lusoria due to incomplete double aortic arch Report of 2 cases Successful surgical treatment) *Bull Soc méd hôp Paris* 68(34-35-36) 1266-1270 1952
- Asherson (N) Dysphagia lusoria with a record of three cases *J Laryng* 66(3) 111-131 1952
- Schmidt (J) Die Arteria lusoria Zusammenfassende Darstellung unter Berücksichtigung eigener Untersuchungen (Arteria lusoria A comprehensive report with reference to personal observations) *Arch Kreis Forsch* 19(1-3) 1-37 1953
- Scott (H W Jr) and Daniel (R A Jr) Tracheal and esophageal compression by congenital vascular anomalies *Laryngoscope* 63(5) 363-378 1953
- Thomsen (G) Vesterdal (J) and Husfeldt (E) Abnormalities of the great vessels (A case of double aortic arch and two cases of anomalous right subclavian artery) *Acta oto-laryng* (Stockholm) 43(suppl 109) 182-188 1953

- Hillemand, Durand et Malpas (P et R) Un cas de mégacolon chez un toxicomane ds paraissant après désintoxication *Soc méd d hôp de Paris* 10 juil 1942
- Hillemand et Viguié (R) A propos de la pathogénie du méga œsophage *Soc méd d hôp de Paris*, 18 juin 1943
- Hillemand (P) Durand (Ch), Duval et Viguié (R) Méga-œsophage, mégabulbe mégacolon chez une toxicomane Désintoxication Guérison *Soc méd d hôp de Paris*, 18 juin 1943
- Baldenweck (K. M.) Un cas de méga dolicho-œsophage ayant probablement subi une opération de Heller *Soc de laryngol d hôp de Paris* 21 juin 1943
- Bariéty (M) Lesobre (R) et Choubrae (P) Méga-œsophage décelé par l'examen radiologique systématique du thorax *Soc méd d hôp de Paris*, 25 juil 1943
- Bergeret Dyskinésie de l'œsophage terminal *Mém Acad de chir*, 20 oct 1943
- Ferrari Lelli (G) Considerazioni sulla patogenesi della dilatazione diffusa dell'esofago *Marginal Otolaryngol*, IV, fasc 2 1943
- Chapelan (M. M.) Contribution à l'étude du méga œsophage idiopathique Thèse Paris 1943
- Hillemand, Servelle, Chénigé et Viguié (R) A propos de la pathogénie et du traitement du méga œsophage *Presse méd* 11 nov 1944
- Juul (A) Sur le cardiospasmé idiopathique et les résultats du traitement par le dilateur de Starck *Acta oto Laryng*, Stockholm, 32, 1, 1944 pp 85-98
- Sprunt (W. H.) et Harill (J. A.) Cardiospasm North Carolina Med J, 5, 1944 p 238
- Bariéty Cord et Lesobre Image pseudo canalaire de la région hilare droite produite par un méga œsophage, valeur sémiologique de l'élargissement global du médiastin *Soc méd d hôp de Paris* 18 mai 1945
- Knolt (H) La question du cardio-spasme *Schweiz med Wochenschr* 75 22 2 juin 1945, pp 477-479
- Guilleminet et Bérard Spasme œsophagien secondaire à une malformation du cardia chez un enfant de 6 ans arrêt des accidents par une opération de Heller *Soc de chir de Lyon* 24 mai 1945
- Scott (W. J. M.) Idiopathic dilatation of the esophagus Differentiation of clinical types and successful operation in intractable cases *Ann Surg* 172, 4 oct 1945 pp 582-605
- Quénu (J) Le méga œsophage *Actual chir*, 1945 Legrand, éd., Paris
- Jensen (V) Cardiospasmé avec excessive dilatation de l'œsophage simulant une tumeur du médiastin *Périod de Méd gén*, Etats scandinaves, no 4, 26 janvier, p 116 in J de chir 67 10-12 1945
- Giampalo L. hypertrophie dite idiopathique des muscles lisses de l'œsophage *L'Informateur méd*, fév 1946
- Delay (J) La narco analyse d'une dysphagie *Presse méd* no 26 8 juin 1946 p 380
- Santy (M) Variétés pathogéniques du méga-œsophage *Rassegna internat di clin e terap* Naples 26 13-14 15-31 juil 1946
- Gaillon (F) Un cas de méga œsophage géant J de méd de Bordeaux et du Sud Ouest 173 15 16 août 1946
- Manguel (M) et Neer (R) Méga œsophage, méga-côlon et cœur *Prensa méd argent* 33, 8 29 nov 1946 pp 2408-2417
- Bensaude (A) et Viguié (R) Méga-œsophage et cancer *Soc de gastro-entéro*, de Paris 9 déc 1946
- Higuer (G) Interprétations pathogéniques des symptômes du cardiospasmé *Bull de la Soc belge d'O R L* no 1 1946
- Mouzon et Nemours Auguste Action du sucre sur l'évacuation du dolicho-méga œsophage *Soc de Gastro-entéro de Paris* 10 mars 1947 in Arch d mal de l'app digest 36 1947
- Nemours Auguste Action de la syncope sur l'évacuation du dolicho-méga-œsophage *Soc de gastro entéro* mars 1947 in Arch d mal de l'app dig 36 1947
- Hillemand (P) Viguié (R) Hartmann et Foix. Les méga-œsophages secondaires *Soc méd d hôp de Paris* 16 mai 1947
- Muller (B) Perron (R) Géniet (F) et Giroux (F) Un cas de méga-œsophage consécutif à une infection à virus neurotrope *Soc méd d hôp de Paris* 16 mai 1947 in Sem d A'p de Paris no 23 21 juin 1947
- Auguste (C) et Paris (J) Un cas de méga œsophage avec syndrome de compression médiastinale *Soc de gastro-entéro de Paris* 16 juin 1947
- Sweet (R.) Dilatation idiopathique de l'œsophage *Surg Clin North Amer* (Massa husetts General Hospital Number) oct 1947 1128-1135
- Mayer (J) Cardiospasmé secondaire diagnostiqué par gastro-œsophagoscopie rétrograd *Soc autrich d'oto-Laryngol*, 2 déc 1947 in Monats fur Ohrenh, mars 1948

- Cuhna (I') Achalasie et méga-œsophage complication et séquelle de la coqueluche *Rev Gastroentérol*, 14 10 1947 pp 693-704
- Loubejac (A) Spasme permanent de l'œsophage distal *Arch Urug de Med, Cir y Especialid Montevideo* 16 2, août in *J de chir*, 63 9 1947
- Sadowski Jeune fille arabe de 17 ans principal signe impossibilité de la déglutition des solides depuis 5 ans *Acta oto-laryng orient* Tel Aviv 3 1-2 1947, pp 26-27
- Jemmu (C.) Hypertrophie musculaire idiopathique de l'œsophage *L Oto rino laringol ital* Bologna 15, 5 1947 pp 348-362
- Camentron et Jammes A propos du méga-œsophage *Ann d oto laryngol* 1947 p 613 in *Concours méd*, 28 nov 1948
- Hillemand Gilbrin, Morin Caronnet et Lapresle Méga cœlon chez une myxœdémateuse *Ann d'oto-laryngol*, 1947 p 614
- Gravano (L.) Méga-œsophage idiopathique *Fl Dia Medica* Buenos Aires 19 12 1947 pp 417-422
- Pinto de Moura et Melho Filho (G de) Méga-œsophage et électrocardiogramme *Rev Bréil d'O R L* Sao Paulo 15, 3 1947 pp 109-111
- Bonadies (A.) Le méga-œsophage *Arch ital d malatt dell appar diger* 13 fasc 2 1947 pp 79 à 126
- Krnyska (S.) D Alambert (G.), Rezendiss et Rango (S.) Infantulisme universel chez un malade porteur d'un méga-cœlon et d'un méga-œsophage *O Hospital* 31 1 1947 pp 21-23
- Szenes (H.) Méga-œsophage et cancer *Mischr Ohrenheil*, 81 1947 pp 100-202
- Godlewski. Méga-dolicho-œsophage *Montpellier méd* 89 5 sept-oct in *J de chir* 63 9 1947
- Euzière Pages Duc Vallat et Latour Méga-œsophage fonctionnel et névralgie méningée au cours d'un zona ostique *Soc des Sc méd et biol de Montpellier et du Languedoc médit* 6 fév 1948
- Dostal (J.) La dilatation idiopathique de l'œsophage *Lekarske Listy Brno* 3 6 15 mars 1948
- Even (M.) et Barré Méga-œsophage Trois cas *Soc franç de pathol resp* 11 avril 1948
- Sénèque (J.) et Roux (M.) Le méga-œsophage *La France méd* no 4 avril 1948
- Umdestock (R.) Sur un cas de méga-œsophage *Soc nat franç de gastro entérol de Paris* 11 mai 1948
- Jammes Cantagnil et Prouzet Un cas de dolicho-méga-œsophage total *Soc de med chir et pharm de Toulouse* juin 1948
- Wooler (G H.) Cardiospasm *Thorax* 3 2 juin 1948 pp 33-70
- Starck (H.) Die Behandlung der Cardiotonischen Oesophagus Dilatation sogen Cardio spasmus *Zschr Lar Rhin Otol* 1 3 aout sept 1948 pp 196-206
- Resano (J H.) Commentaires sur des œsophagopathies de diagnostic difficile *Ann de O R L del Uruguay* 16 4 pp 287-305 In *Annales d oto laryngol* 65 11 nov 1948
- Mayer (J.) Sekundärer Kardiospasmus diagnostiziert durch retrograde Gastro œsophago skopie *Mischr Ohrenh* 82 3 1948 p 137
- Hillemand (P.) Beau (H.) Womant et Sonea A propos des méga splanchnies digestives fonctionnelles en rapport avec une atteinte du système nerveux central *Bull et mém Soc méd d hop de Paris* extrait des nos 32 et 33 1948 p 1096
- Filloux (J.) Contribution à l'étude du méga-œsophage *Thèse Paris* 1948
- Foux (H.) Contribution à l'étude des méga-œsophages secondaires *Thèse Paris* 1948
- Clerc (P.) Méga-œsophage *Actualités O R L* 1948 Librairie Arnette Paris
- Even et Barré Méga-œsophages décelés sans opacification préalable par l'examen radio logique thoracique *J franç méd chir thor* 2 5 1948 p 467
- Fontana (R.) Considerazioni sul megaeosofago idiopatico e sulle dilatazioni esofago diffuse secondarie a stenosi *Atti Chn O R L di Siena* in *Suppl dell O R L Ital* 1948
- Castagnari (G.) Un caso di megaeosofago a sintomatologia muta *Arch ital mal app dig* 14 4 1948 p 344
- Bachm Kardiospasmus *Med Klin* 1948 pp 69-70
- Szenes (H.) Contribution à la pathogénie et à la thérapie du spasme du cardia *Wien klin Wschr* 1948 pp 144-147
- Riccabona (A V.) Idiopathisches Oesophagus Dilatation *Mischr Ohrenh* 82 2 1948 pp 88-91
- Som (A L.) Cardiospasm chez l'enfant *Indian J Pediatr* Calcutta 15 58 1948 pp 69-72
- Razemon et Delvas A propos du méga-œsophage *Soc de chir de Lille* 14 janv 1949
- Lehoucq (J M.) Méga-œsophage agénésie de la première cote gauche et bronchiectasie gauche *Soc de pathologie respiratoire* 13 mars 1949

- Prouzet et Layssol Les aspects pseudo lésionnels du parenchyme pulmonaire dus au méga-œsophage. *Soc de méd, chir et pharm de Toulouse* avril 1949
- Nemours Auguste L'extrémité inférieure de l'œsophage dans le dolicho méga-œsophage. *Soc nat franç de gastro entérol*, 13 juin 1949
- St O Aylett Un cas avancé de méga-œsophage du au cardiospasme. *Brit J Surg*, Londres, 36 143 juil 1949
- Benassi (E) Dyschalasie du cardia et dystonies gastro intestinales. *Minerva med* Turin 40 38 4 août 1949, pp 133-137
- Evans (G) Les cardiospasmes. *The Practitioner*, Londres, 163 975, sept 1949, pp 173-256
- Kramer (P) et Ingelfinger Le cardiospasme trouble généralisé de la motilité de l'œsophage. *Am J Med*, 7 2 août 1949, pp 174-179
- Alvarez (W C) Une explication simple du cardio spasme et de la maladie de Hirschsprung. *Gastro Enterology*, 13, 5, nov 1949 pp 422-429
- Wanke (R) et Schuttemeyer Remarques critiques sur le cardio spasme. *Der Chir*, Berlin 20 6, juin 1949 pp 266-273
- Wolff (S) et Almy (T P) Observations expérimentales sur le spasme du cardia chez l'homme. *Gastro Enterology* Baltimore 13, 5, nov 1949 pp 401-422
- Turpin Morin (G) Lemoine (J M) et Piton Image claire cervicale traduction radiographique d'un méga-œsophage groupement dysmorphique particulier. *J franç de méd et de chir thor*, Paris 3, 5, 1949
- Udaonto (C B) Maissa (P A) et Di Alotto (V) Contribution à l'étude de la pathologie fonctionnelle de l'œsophage. *Prensa méd argent* Buenos-Aires, 36 4 1949 pp 173-178
- Magonet (A P) Cardiospasm. *Lancet*, 1, 1949 p 629
- Wanke Kritische Bemerkungen zum sog Kardiospasmus. *Klin Woch* 1949, 184
- Schuhemeyer Sogenannter Kardiospasmus und stenosierende Oesophagitis. *Klin Wochr* 1949 184
- Toback Kardiospasmus und Kardiadilatation nach Starck. *H N O* 1, 1949, p 281
- Hillemand (P) et Lecoœur (Mme) La mégasplanchnic digestive chez les toxicomanes. *Bull et mém de la Soc méd d hôp de Paris* extrait des nos 5 et 6, 1949, p 143
- Lemoine (J M) Méga-œsophage à image claire agénésie de la 1re côte gauche et bronchectasie gauche. *J franç de méd et de chir thor*, Paris 3, 4, 1949
- Nese (G) L'achalasie œsophagienne. *Tidsskr d f norske lægefor* 69, 4, 1 fév 1949
- Da Costa Quinta (A) Dilatations bénignes diffuses de l'œsophage. Considérations cliniques et endoscopiques. *Clin Higien e Hidrologia* Lisboa 15 1 1949 pp 16-23
- Bertrand (L) Le méga colon dans la maladie de Steinert. *Rev neurol*, 81, no 6 1949
- Pruvost Meyer (A) et Chétien Le méga-œsophage, découverte d'examen radiologique systématique des poumons. *Soc méd d hôp de Paris* 24 fév 1950, in *J de chir* 66 6-7 juin juillet 1950
- Huguenin (A) Charles (F) et Zevaco (P) Méga-œsophage sur téléthorax standard sans opacification préalable. *Soc méd d hôp d Alger*, 8 mars 1950
- Hubbard (T B Jr) et Leven (N L) La dilatation œsophagienne une contre indication au cathéter déglutit et une méthode alternative. *Surgery*, 7, 1, janv 1950
- Santy et Michaud Méga-œsophage. *Soc de chir de Lyon*, 1 juin 1950 in *Presse méd*, no 42 1 juillet 1950
- Nemours Auguste Etude de la partie terminale de l'œsophage dans le dolicho-méga-œsophage. *Sem d hôp de Paris* 14 mars 1950
- Simonetti (C) Achalasia of the cardia due to vitamin B deficiency. *Annali ital di chir* (Bologna) 24(3-4) 137-145 1947
- Wolf (S) and Almy (T P) Experimental observations on cardiospasm in man. *Gastroenterology*, 13 401-421, 1949
- Staudacher (V E.) and Belli (L.) La motilité esofagea nel megaesofago cardiospastico e sperimentale (Rilevazioni fisiopatologiche) (Esophageal motility in experimental cardiospastic megaesophagus). *Arch ital mal appar dig* 16(5) 383-412 1950
- Eberle (J) Zur Ätiologie der idiopathischen Oesophagusdilatation (The etiology of idiopathic esophageal dilatation). *Wien klin Wochr*, 1951 63(4) 67 1951
- Hillemand (P) Hepp (J) and Viguet (R) Megaesophage et ulcère de la petite courbure Anastomose œsogastrique sus diaphragmatique. *Gastrorectomie secondaire pour hématoémèse grave* (Mega-esophagus and ulcer of the lesser curvature Subdiaphragmatic esophagogastric anastomosis Secondary gastrectomy for severe hematemesis). *Arch mal appar dig* 40(7-8) 891-895 1951

- Santy (P) and Michaud (P) Dyskinésie œsophagienne (Dyskinesia of the esophagus) *Arch mal appar dig*, 1951 suppl 5 pp 166-184
- Santy (P) and Michaud (P) Cardiospasme et cancer de l'œsophage (Cardiospasm and cancer of the esophagus) *Lyon chir*, 46(1) 105-107 1951
- Soler Roig (J) Disquinesias esofágicas (Esophageal dyskinesia) *Boll Soc Piemont chir* 73(7) 473-482 1953
- Slesinger (M H) Steinberg (H) and Almy (T P) Disturbance of esophageal motility in cardiospasm studies on autonomic stimulation and autonomic blockade of human esophagus including cardia *Gastroenterology* 25 333-348 1953
- Barrett (N R) Achalasia thoughts concerning the ætiology *Ann Roy Coll Surg England* 12(6) 391-402 1953
- Corelli (D) L'infiltrazione con novocaina a lento assorbimento (proctocaine) dell'esofago epicardio-cardiale contributo sperimentale allo studio della patogenesi del mega-esofago essenziale (Slow absorption procaine (proctocaine) infiltration of the epicardial and cardiac parts of the esophagus experimental contribution on the pathogenesis of essential mega esophagus) *Arch chir (Torace)* 8(3) 571-588 1954
- Taber (R E) and Ehrenhaft (J L) Esophageal motility in cardiospasm *Arch Surg* 69(2) 154-166 1954
- Nemur (P Jr) and Hawthorne (H R) Physiologic basis for utilization of esophago-cardio-myotomy in the treatment of achalasia *J Thoracic Surg* 28(3) 247-258 1954
- Olsen (A M) Ellis (F H Jr) and Creamer (B) Cardiospasm (Achalasia of the Cardia) *Am J Surg* 93(2) 299-307 1957
- Deloyers (L) Cordier (R) and Duprez (A) A new approach to the physiology of so-called cardiospasm *Ann Surg* 146(2) 167-177 1957

TREATMENT OF MEGA ESOPHAGUS

- Santy (P) Opération de Heller pour spasme de l'extrémité inférieure de l'œsophage *Soc de chir de Lyon* 4 mai 1939
- Healy (C A) Le traitement chirurgical du cardio-spasme inaméliorable *XIII Reunion de l'Assoc amér de Broncho-Œsophagol* 5 juin 1940
- Ledoux Lebard et Nemours Action des nitrites sur le cardio-spasme du mega-œsophage *Acad de méd* nov 1940
- Sénéque (J) Cardio-spasme de l'œsophage Opération de Heller Résultat *Acad de chir* 4 déc. 1940
- Brauer Sur les sténoses œsophagiennes dites idiopathiques et les mega-œsophages *Acad de chir* 11 déc. 1940
- Desplas (B) et Aimé (P) A propos des sténoses du cardia avec mega-œsophage secondaire *Mém Acad de chir* 15 janv 1941
- Santy Ballivet et Bérard Méga-œsophage et cardio-spasme *Soc de chir de Lyon* 13 mars 1941
- Bérard Ballivet Un nouveau traitement chirurgical du cardio-spasme essentiel *Soc O R L Lyon* avril 1941
- Stinson (W D) Les effets du chlorure de thiamine sur le cardio spasme *Ann of O R L* 50 3 sept. 1941 pp 898-903
- Luras Méga-œsophage traité par l'opération de Heller *Soc de chir d'Alger* 21 nov 1941
- Louche (P) Traitement des sténoses cardio-œsophagiennes essentielles par l'opération de Heller *Thèse Paris* 1941
- Patel et Charbon Méga-œsophage opération de Heller *Soc de chir de Lyon* 23 avril 1942
- Hepp (J) Léger (J) et Marzet Réflexions à propos de cinq observations de mega-œsophage traité par l'opération de Heller *Sem d hop de Paris* août 1942 pp 219-225
- Santy (P) Bérard (M) Ballivet (M) et Magnin (F) *Traitement chirurgical du mega œsophage et cardio-spasme* 1 vol de 50 p avec 20 fig Vigot édit 1942
- Santy (P) Bérard (M) Ballivet (M) et Magnin (F) Traitement chirurgical du syndrome mega-œsophage et cardio-spasme *Presse méd* 20 mars 1943 no 11 pp 134-135
- Santy (P) Bérard (M) et Ballivet A propos de 15 observations de mega-œsophages traitées chirurgicalement *Lyon chir* 38 2 mars avril 1943 pp 115-123
- Santy (M) Traitement actuel du mega-œsophage par cardio-spasme *Mém Acad de chir* 9 juin 1943
- Santy (P) Bérard (M) Traitement du mega œsophage D'après 27 interventions *Acad de chir* 17 juin 1943 p 69
- Hillemand Chérigie Servelle et Vigné A propos du traitement du mega-œsophage *Soc méd d hop de Paris* 18 juin 1943

- Stefaniu (A), Pitschak (C) et Anaste Sescu (I) Cardiospasme chronique avec dilatation œsophagienne Cardio myotomie Guérison *Rev romana de oto-rino-laryngol* oct-déc. 1943
- Jeanneney, Magendie Tingaud et Bouet Un cas de méga-œsophage splanchnectomie et opération de Heller en un temps *Soc de chir de Bordeaux et du Sud Ouest* 10 fév 1944
- Bourget Cardio spasme avec échec de la dilatation et de l'infiltration des nerfs lombaires Opération de Heller Guérison *Soc de laryngol d'hôp de Paris* 21 fév 1944, p 269
- Hepp (J) Données actuelles sur le traitement chirurgical du méga œsophage *Rev de chir Paris* no 9-10 sept-oct 1944
- Artaud Dilatation idiopathique de l'œsophage, opération de Heller guérison *Soc de chir de Marseille*, 8 janv 1945
- Clagett (O T) Moersch (H J) et Fischer (A) Esophagogastrostomy in the treatment of cardiospasm *Surg Gynec & Obst*, 81, 4 oct 1945, pp 440-445
- Weyder Action de la nitro glycérine sur le cardio spasme *Acta med Scand*, 123 1 1945 pp 12-25
- Patel Cardio-spasme, libération simple de la partie inférieure de l'œsophage *J de Chir*, 61, 1-3, 1945
- Hillemand Idrac et Cazeilles Cardio spasme opération de Heller *Sem méd d hôp de Paris*, 1 mars 1946, pp 147-149
- Fontaine, Forster et Stefanini Le méga-œsophage Résultats de la splanchnectomie *Lyon chir* 41, 3, mai juin 1946 p 289
- Bell (H) The treatment of cardiospasm by esophagogastrostomy *Surgery* 20 1, juil 1946 pp 104-116
- Grinson (S S) Reeves (R J) Trent (J C) et Wilson (A D) The treatment of patients with achalasia by esophagogastrostomy *Surgery* 20 1 juil 1946 pp 94-103
- Magendie et Tingaud Double opération en un temps pour méga œsophage (opération de Heller et splanchnectomie gauche) *Presse méd*, 54 37, 10 août 1946
- Schiebel (H M) Treatment of esophageal achalasia or cardiospasm Report of four patients treated surgically *Surgery*, 20 4 oct 1946 pp 458-570
- Lanzara (A) La divulsion per orale et la myotomie extra muqueuse dans le traitement du soi disant cardio spasme *Il Polichin*, 53, 11-12 15 nov 15 déc 1946 pp 235-243
- Cuyppers (I) Traitement des cardio spasmes soi disant primitifs *Monat sur Kinderheilt Berlin* 96 1 1946 pp 30-32
- Savinykh (A G) Esophagogastrostomy for Cardiospasm *Am Rev Soviet Med* 3, 297 1946
- Tucker (G) Dilatateur pneumatique à mercure pour le traitement du cardiospasme *Ann Oto-Laryngol* 13 9-10, 1946 p 467
- LongGrove et Mac Millian Traitement chirurgical de l'achalasia de l'œsophage *Quart Rev O R L*, mars 1947
- Ceranke (P) La dilatation idiopathique de l'œsophage et son traitement par les corps nutritifs *Wien klin Wochenschr*, 59 41 17 oct 1947, pp 681-683
- Morone (C) Sur deux cas d'achalasia œsophagienne opérée par cardiomyotomie de Heller *Giornale ital di chir Nap'les* 3 10 oct 1947
- Cattan (R) Frumusan (P) et Attal (C) Un cas d méga-œsophage traité par le bromure d'acétyl H méthylcholine *Sem d hôp de Paris*, no 48, 28 déc 1947
- Broyles Nouveau dilateur œsophagien *O R L internat* déc 1947, p 191
- Hanke (H) Ueber den Kardiospasmus und seine Behandlung *Archiv f. d. Naturforsch* 1947 pp 1057-1061
- Lanzara (A) Contributo all interpretazione ed alla terapia del cardiospasmio *Polichinco*, 54 1947 p 1299
- Lanzara (A) Le cardio spasme Essai de définition clinique et pathogénique Thérapeutique chirurgicale *Arch di chir del Torace* 1 1947, pp 55-159
- Nagel (G W) et Menkh (J) Opération transthoracique pour méga œsophage *Ann d oto-laryngol* 1947 p 615
- Pavia (J) Méga-œsophage traité par dilatation aux bougies de Hegar du cardia voie intra gastrique *Arch Urug de med, cir y especialid Montevideo* 16, 5, nov In *J de chir*, 63 9 1947
- Kay (E) Le traitement chirurgical du cardiospasme *Ann Surg* 127 1, janv 1948
- Ilotegher (A) et Biondetti (P) Suites cliniques et radiologiques à distance de l'opération de Heller pour méga-œsophage *Minerva chir* 3 2 fév 1948 pp 65 à 78
- Gill Dan (C) et Child (Ch G) Esophagogastrostomy in the treatment of cardiospasm *Surgery* 23 3 mars 1948 pp 571-583
- Kay (E B) Surgical treatment of cardiospasm *Ann Surg*, 127, 1948 pp 34-39

- Roux (M) Diagnostic clinique et indications thérapeutiques du méga œsophage *Presse méd* suppl au no 35 du 16 juin 1948
- Joyeux (R) Méga-œsophage Opération de Heller Guérison *Soc des Sc méd* Montpellier 9 juil 1948
- Gaujoux (A.) et Fabre (L.) Deux cas de méga œsophage opérés *Sem d hôp de Paris*, no 54 18 juil 1948
- Zerbini (E.) Résection transthoracique du cardia avec œsophago gastrostomie dans les cas de méga-œsophage non guéris par la cardio myotomie de Heller *Rev de cir de São Paulo* 14, 2 sept.-oct 1948
- Swynghedauw et Salembier Méga œsophage traité par opération de Heller *Soc de Chir de Lille* 10 déc. 1948
- Fontana (L.) Sur le traitement chirurgical selon Heller du méga-œsophage et cardio spasme *Ann ital di chir*, Bologne, 25, fasc 12 déc 1948 pp 845-855
- Bombi (G) Contributo al trattamento neurochirurgico del megaesofago *Arch ital mal app dig*, 14 4 1948 pp 351-356
- Mission (J) Remarques sur le méga-œsophage et le traitement par la sonde à mercure *Acta gastro-enterol belgica*, Bruxelles 2, 2 1948 pp 101-104
- Stark (H) Die Behandlung des Cardiotonischen Oesophagus Dilatation sogen Cardio spasmus *Z Laryng U S W*, 1, 1948 Op 196-206
- Isaac (J E.) Treatment of esophageal achalasia or cardiospasm report of a case treated surgically after bougienage failed *Manitoba M Rev* 28 1948 p 463
- Pieri (G) La cura chirurgica del cardiospasma *Arch ital mal app dig* 14 4 1948 pp 326-336
- Frazao (M) et Saldanha (A) Sur le méga-œsophage à propos de deux cas opérés *Gazeta Med Portug* Lisbonne, 2 1, janvier avril 1949
- Lanzara (A.) Interventions radicales par voie transthoracique dans le traitement du cardio spasme *Il Policlin* 56 7, 14 fév 1949 pp 198-203
- Dubourg (G) Réflexions sur l'opération de Heller dans 17 cas de méga œsophage *Soc nat franç de gastro-enterol*, 13 mars 1949
- Patel (J) et Binet (J P) Chirurgie du dolicho-méga-œsophage œsophago gastrostomie de nécessité ou de principe? *Presse méd* 23 mars 1949
- Costantini (H) et Leca (A.) A propos de la section extra muqueuse des fibres musculaires du cardia dans la cure radicale du cardio-spasme *Rev chir* 87 3-4 mars avril 1949 pp 80-87
- Nuber Œsophagocardioplastie pour achalasia du cardia *Nederl tijdschr voor geneesk* Haarlem 93 15, 9 avril 1949
- Cottalorda. Méga-œsophage Œsophago-gastrostomie transthoracique *Soc de chir de Marseille* 25 avril 1949
- Bocca (E.) Action de quelques médicaments sur le tableau clinique et radiologique du cardio spasme *Minerva med* Turin 1 23 12 mai 1949 pp 684-690
- Paliard Plauchu Ollagner et Coppéré Méga-œsophage chez une hypothyroïdienne aggravation de la dysphagie par le traitement thyroïdien Amélioration temporaire par la yohimbine guérison par l'opération de Heller *Lyon méd* 81 20 16 mai 1949
- Courty (L.) Un cas de méga-œsophage Opération de Heller Guérison *Soc méd et anatomo clin de Lille* 27 mai 1949
- D Allaines Lortat Jacob et Gauchy Traitement du méga-œsophage *Acad de chir* 1er juin 1949
- Hepp A propos du traitement du méga-œsophage *Acad de chir* 1 juin 1949
- Orvier (CL) Albort (Guy) et Berthet (G) L'œsophago-gastrostomie dans le traitement du dolicho-méga-œsophage *Presse méd* no 36 1 juin 1949
- Redon (H) et Lacoste (J) Deux échecs de l'opération de Heller traités avec succès par la cardioplastie *Acad de chir* 1 juin 1949
- Monod (R.) et Germain. Œso-gastrostomie par voie abdominale pour méga œsophage *Acad de chir* 8 juin 1949
- Bolot (P) Chailiol (J) et Nègre (A.) Trois cas de cardio-spasme œsophagien traités par cardiectomie extra muqueuse de Heller associée à la neurotomie sous-diaphragmatique des deux pneumogastriques *Acad de chir* in *Presse méd* 2 juil 1949 no 43
- Delannoy A propos du traitement du méga-œsophage par l'opération de Heller *Acad de chir* 13 juil. 1949
- Ferrer (E. A.) Trois cents cas d'achalasia du cardia traités par dilatation *IV Congrès intern d'oto-laryngol.*, Londres 18-23 juil 1949
- Courty (L.) Méga-œsophage avec sténose hypertrophique du cardia *Lille chir* 4 22, juil.-août 1949

- Kramer (P) et Ingelfinger (J) Le cardiospasme désordre généralisé de la motilité œsophagienne *Am J Med* no 2, août 1949
- Barnard (A) et Bataille (A) Résultat thérapeutique d'un méga œsophage sept ans après une opération de Heller *Soc méd et anat lin de Lille*, 26 oct 1949
- Becquet (R) et Bataille (A) Une nouvelle épreuve pharmacodynamique dans le dolicho-méga œsophage Essai d'un traitement médical *Soc méd et anat clin de Lille*, 26 oct 1949
- Barrett (N R) et Franklin (R H) Au sujet des mauvais résultats lointains de certaines interventions dans le traitement du cardio spasme *Brit J Surg*, 37, 146, oct 1949, pp 194-202
- Charrier (J), Gautier (Ch) et Tedesco (Mme) Sur un cas de méga œsophage monstrueux traité par œsophago gastrostomie *Soc nat franç de gastro-entérol*, 14 nov 1949
- Santy et Michaud Le traitement du méga œsophage et cardio spasme, à propos de 94 cas d'opération de Heller *A cad de chir*, 30 nov 1949
- Donche (R) A propos de 11 cas de méga œsophage traités par l'opération de Heller *Acta gastro entérol bel*, 12, 11, nov 1949
- Santy A propos du traitement chirurgical du méga œsophage *Acad de chir*, 7 déc 1949 in *Presse méd* no 79, 17 déc 1949
- Allison (P R) Obstruction de la jonction gastro œsophagienne *The Lancet*, London 2 3 1949, pp 91-94
- Maingot (R) Traitement chirurgical du cardio spasme *Postgraduate Med* 5 5 1949, pp 351-360
- Salzer (G) Traitement opératoire du cardio spasme *Wien klin Wochenschr* 1949, p 79
- Cottalorda Pedimetre et Devin Le problème du traitement chirurgical du méga-œsophage *Marseille chir* 2, 1 janv fév 1950
- Fontaine A propos des méga œsophages *Acad de chir*, 1 fév 1950
- Lortat Jacob Inconvénients de l'œsophago gastrostomie dans les échecs de l'opération de Heller *Soc nat de gastro entérol* 17 fév 1950
- Mernel (E) Comment concevoir et traiter actuellement le méga œsophage *Congrès méd* 25 mars 1950 p 895
- Fontaine (R) La cardio œsophagotomie extra muqueuse élargie par voie intrathoracique et associée à la résection des splanchnique et sympathique gauches dans le traitement du méga œsophage *Acad de chir*, 1er fév 1950
- Bernard (A) et Bataille (A) Le résultat thérapeutique d'un méga œsophage après une opération de Heller *J de Soc méd Lille*, 68, 4 5 fév 1950
- Pupple (I D) Le rôle de la motilité de l'œsophage dans le traitement chirurgical du méga œsophage *J Thoracic Surg*, 19 3 mars 1950
- Baerema Méga œsophage *Ned tijdschr voor geneesk*, an 94, no 9 mars 1950
- Swynghedauw et Salembier Méga œsophage traité par opération de Heller, *Lille chir*, mai juin 1950
- Lovgue (J) Notions actuelles sur le traitement du méga œsophage *Sem d hôp de Paris*, 18 juin 1950
- Hillemand, Vigué Bernard (H J) et Decaudaveine Sur le traitement chirurgical des méga œsophages *Acad de chir*, 25 oct 1950
- Vinson (P P) Diagnosis and treatment of cardiospasm *Postgrad Med* 3(1) 13-18 1948
- Hermunghaus (H) Die kardionische Ösophagusdilatation im Kindesalter (Cardionosis dilatation of the esophagus in children) *Mischr Kinderheilk* 97(1) 1949
- Wulff (H B) and Malm (A) Transpleural cardioplasty in achalasia Operation results and sequelae *Thorax*, London 4(4) 243-250 1949
- Delovers (L) Comment traiter chirurgicalement les échecs de l'opération de Heller pour cardiospasme Proposition et discussion d'une technique (Suggestions for the surgical treatment of failures with Heller's operation for cardiospasm Discussion of technique) *Acta gastro ent bel* 13(9-10) 949-960 1950
- Barrett (N R) et al Discussion on treatment of achalasia of the cardia *Proc Roy Soc Med* 43(6) 421-434 1950
- Fontaine (R) and Grosse (A) La cardio-œsophagotomie extra muqueuse élargie par voie intrathoracique et associée à la résection des splanchnique et sympathique dorsaux gauches dans le traitement du méga-œsophage A propos de 6 cas personnels (Larg extra mucous cardio-esophagotomy combined with left sided sympathectomy and splanchnicotomy for mega-esophagus Report of 6 cases) *Mém de l'Acad de chir Paris* 76(4-5) 216-224 1950
- Malm (A) Cardioplasty in the surgical treatment of achalasia of the esophagus A critical

- review founded on experimental studies *Scand J clin lab invest* 3 (suppl 1) 7-89, 1951
- Scott (W J M) and Emerson (G L) The choice of treatment in idiopathic dilatation of the esophagus *Acta Gastroent*, 18(4) 257-266 1951
- Kay (E B) Observations as to the etiology and treatment of achalasia of the esophagus *J Thoracic Surg* 22(3) 254-270 1951
- Hillemand (P) Viguié (R) Bernard (H) and Decaudaveine Le traitement des méga-œsophages (Treatment of mega-esophagus) *Sem d H p de Paris* 77(88) 3503-3506 1951
- Olsen (A M) Harrington (S W) Morsch (H J) and Andersen (H A) The treatment of cardiospasm Analysis of a twelve year experience *J Thoracic Surg* 22(2) 164-187 1951
- Lanzara (A) Revisione critica del problema terapeutico del così detto cardiospasm (Critical review of the treatment of so-called cardiospasm) *Arch chir (Torace)* 6(4) 525-553 1952
- Wulff (H B) and Malm (A) Considerations and treatment of achalasia of the esophagus *Acta chir Scand* 103(6) 445-458 1952
- Guglielmini (G) Actis Dato (A) and Scala (D) Terapia chirurgica del cardiospasm valutazione dei risultati a distanza di tempo dall intervento (Surgical treatment of cardiospasm a study of the late results of the operations) *Arch Sci Med* 100(5) 390-402 1955
- Sweet (R H) Surgical treatment of achalasia of the esophagus *New England J Med* 254(3) 87-95 1956
- Santy (P) Michaud (P) and Lastreille (R) Le traitement du mégaœsophage par l'opération de Heller Résultats de 168 interventions (Treatment of mega esophagus by Heller's operation Results of 168 operations) *Lyon chir* 51(5) 513-522 1956

Chapter 10 Atony and Paralysis

- Bouchet et Debaun. Trois cas de botulisme Inerte pharyngée dans le botulisme *Radios Oto-rhino-laryngol internat* an 32 t 28 no 3 mars 1944
- Alajouanine (Th) Thurel (R.) et Durupt (L) Le botulisme maladie nerveuse *Presse med* 20 janv 1945
- Michon (P) Heuilly (F) Lesure (J) et Delestre (J) A propos du botulisme Etude de quelques troubles humoraux et de la dysphagie Le moulage passif de l'hypopharynx *Soc méd hop de Paris* 28 juin 1946 pp 377-380
- Moumier Kuhn Monnet Moinecourt et Favre Cilly Les troubles de la déglutition au cours du botulisme Dysphagie simple et paralysie de l'œsophage *J med Lyon* 5 sept 1946 pp 629-632
- Dereux Baude et Lefebvre (L) Botulisme dysphagie sans paralysie œsophagienne *J d Sc méd de Lille* 10 nov 1946
- Potereau (L J) Les paralysies gastro-œsophagiennes au cours de la diphtérie *Thèse Paris* 1946
- Maisonobe (L) Deux cas de paralysie de l'œsophage d'origine diphtérique Syndrome du cinquantième jour *Ann Oto-Laryng* 13 7-8 1946 pp 365-366
- Michon (P) Paralysie diphtérique œsophagienne isolée *Rev méd de Nancy* 15 fév 1947
- Maisonobe et Lemarié Deux cas de paralysie de l'œsophage dont une diphtérique *O R L internat* déc 1947 p 12
- Michon (P) Herbeval (R) Faivre (G) et Andlauer Paralysie de l'œsophage au cours d'un syndrome malin tardif d'une diphtérie *Ann d oto laryng* 1947 p 415
- Neuhauser (E H D) and Berenberg (W) Cardio-esophageal relaxation as cause of vomiting in infants *Radiology* 48 480-483 1947
- Kaplan (S) Paralysis of deglutition a post poliomyelitis complication treated by section of the cricopharyngeus muscle *Ann Surg* 133(4) 572-573 1951

Chapter 11 Diverticula

- Cavina Diverticule de l'œsophage cervical résection en un temps guérison de plus de trois ans *Il Policlinico Rome* 44 38 20 sept 1937
- Guarnaccia Micro diverticulose kystique suppurée de l'œsophage *Oto-rhino-laryngol ital* 10 5 sept 1940 ■ 463
- Nemours Auguste Aspect diverticulaire épiphrenique de décubitus *Soc de Gastro-Enterol de Paris* 3 janv 1941 in *Arch des mal de l'app dig* 30 1-2 janv fév 1941
- Israel (S) Une méthode pour faciliter la promptre reconnaissance du diverticule œsophagien au moment de l'opération *Surgery* ■ 5 mai 1941

- Varela Fuentes et Garcia Capurro Diverticulose de l'œsophage 12 cas *Arch urug de med, cir y especialid*, Montevideo, 19 1, juil 1941
- Graumann. Diverticules de l'œsophage et carcinome du diverticule *Zentralbl für Chir* 68, 3 17 janv 1942
- Chiappe Recherches expérimentales sur la pathogénie des diverticules œsophagiens *Rec de laryngol, otol rhin*, mars 1942 p 95
- Baldenweck (M) Diverticule de l'œsophage thoracique Paralysie récurrentielle gauche *Soc de laryngol d hôp de Paris* séance du 21 juin 1943
- Bouchet (M) et André (P) Diverticule intrathoracique de l'œsophage *Soc de laryngol d hôp de Paris* 21 juin 1943 in *Ann d oto laryngol* no 7, 1944-1945 juil août sept
- Trial (R) et Marot (R) Double diverticule de l'œsophage *Montpellier méd* n 5, 1943
- Santv Bérard et Clerf Dix observations de diverticules de l'œsophage cervical traités chirurgicalement *Soc de chir de Lyon*, 3-6 juin 1944
- Monod (R) Diverticule sus-diaphragmatique de l'œsophage ouvert dans les bronches et compuqué d'abcès du poumon *Acad de chir* 20 déc. 1944
- Santy (P) Ravault (P) Bérard (M) et Roch Diverticule œsophagien et suppuration pulmonaire *J de méd de Lyon*, no 598 5 déc 1944, pp 821-825
- Gaveau (J) Diverticule de l'œsophage et suppuration pulmonaire *Thèse Lyon*, 1944
- Aubin (A) et Joubin Pathogénie et traitement des diverticules pharyngo-œsophagiens *Ann d'oto-laryngol* 12, 10 1944-1945 avril juin
- Aubin (A) et Joubin. La fable du triangle de Lamer et le siège des diverticules de pulsion pharyngiens *Ann d'oto-laryngol* 12 10 1944-1945 avril juin.
- Santy et Bérard Diverticules épiphreniques et méga-œsophage *Soc de chir de Lyon* 40 2 1945 p 258
- Fscat (M) et Laval (R) Diverticule de l'œsophage et méga pharynx Diverticulopeut complétée par œsophagotomie extra muqueuse *Soc de chir de Toulouse in Presse méd* 27 janv 1945 pp 49-51
- Ludin (Max) Le diverticule œsophagien épibronchique de Lutgert *Schweiz med Wochenschr*, Bale, no 6 10 fev 1945 p 126
- Alder Sur la production des diverticules œsophagiens *Schweiz med Wochenschr* Bale no 1 6 janv 1945 p 126
- Morley (J) Diverticule pharyngien *Manchester Med Soc*, 4 avril 1945
- Harrington (S W) Diverticules de pulsion de l'hypo pharynx et de la jonction pharyngo-œsophagienne *Surgery* 18, 1 juil 1945, pp 66-81
- Drouet Grimaud et Rousseaux. Diverticule épiphrenique de l'œsophage Intervention chirurgicale *Soc de laryng d hôp de Paris* 15 oct 1945
- De Toma (Emmanuelle) Les diverticules dits épiphreniques de l'œsophage *Thèse Paris* 1945
- Melamed (A) et Zimmerman (B M) Fistule œsophago-bronchique à travers un diverticule œsophagien *Am J Roentg & Radiother*, janv 1946
- Harrington (S W) Pulsion diverticula of the hyphopharynx at the pharyngo-œsophageal junction. *Surg Gynec & Obst*, fév 1946
- Welt (M) Intérêt de l'extérésection préalable dans le traitement de certains diverticules cervicaux de l'œsophage *Acad de chir* 5 juin 1946, in *Presse méd* 15 juin 1946
- Moure (P) Radiothérapie d'un diverticule de l'œsophage *Acad de chir*, 5 juin 1946
- Lahey (F H) Un cas de diverticule œsophagien intrapleurale *Laher Clin Bull*, juil 1946
- Delannoy et Vandecasteele Deux cas de diverticules pharyngo-œsophagien traités par la résection en un temps *Soc de chir de Lille* 14 juin 1946 in *Presse méd*, 28 sept 1946
- Janes (R M) Diverticules de l'œsophage thoracique inférieur *Ann Surg*, 124, 4 oct 1946 pp 637-652
- Lahey (F H) Diverticule pharyngo-œsophagien son traitement et ses complications *Ann Surg* 124 4 oct 1946 pp 637-642
- Escat Traitement chirurgical des diverticules de l'œsophage Avantages de la résection en un temps *Soc méd chir et pharm de Toulouse* nov 1946
- Calvet (J) et Claux (J) Diverticule de l'œsophage Etude post-opératoire de la déglutition *Soc de chir de Toulouse*, 20 déc 1946, in *Presse méd* 7 juin 1947
- Fontaine (R) Oswald (A) et Watter (P) Diverticule géant de l'œsophage cervical inférieur à développement médiastinal Résection en un temps après gastrotomie Guérison *Rev de chir* 65 5-6 1946 pp 186-192
- Ballivet. Diverticule de l'œsophage cervical chez un vieillard Ablation en un temps *Mém Acad chir Paris* 72 27-28 1946 pp 477-480
- Debray (Ch) et Le Canuet (R) Pseudo-tumeur thoracique par diverticule épiphrenique *Sem hôp Paris* 21 juin 1946

- Bourgeois (Robert) Volumineux diverticule de l'œsophage réséqué en un temps *Ann d oto laryngol* 13 5-6 1946
- Sweet (R. H.) Diverticule de pulsion de la jonction pharyngo œsophagienne technique de l'opération en un temps *Ann Surg* 125 1 janv 1947, pp 41-48
- McNealy (R. W.) et Glassman (J. A.) Résection en un temps d'un diverticule pharyngo œsophagien *Surgery* 21, 4 avril 1947 pp 470-475
- McNealy (R. W.) et Glassman (J. A.) Diverticule épiphyrénique de l'œsophage traité par diverticulo-gastrostomie sous diaphragmatique *J Internat Coll of Surgeons* 10 3 mai juin 1947 pp 268-271
- Bourgeois (A.) et Siguié (A.) Volumineux diverticule de l'œsophage thoracique *Soc d oto laryngologie de Paris* 16 juin 1947
- Mac Charles (M. R.) Pharyngeal diverticula *Laval méd* 12 6 juin 1947 pp 333-335
- King (B. T.) Nouvelles conceptions sur l'étiologie et le traitement des diverticules de l'œsophage *Surg Gynec & Obst* 85 1 juillet 1947 pp 93-97
- Ramos Mejia Diverticule de l'œsophage *Prensa med argent* Buenos Aires 34 36 5 sept 1947
- Wheeler (D.) Les diverticules non ulcéreux de l'œsophage de l'estomac et du duodénum *Radiology* 49 4, oct 1947 pp 476-481
- Judet et Clerf Un cas de diverticule épiphyrénique opéré *Soc de laryngol d hôp de Paris* 15 déc 1947
- Melamed (A.) et Walker (L.) Un cas de diverticule pharyngo œsophagien intramural disséquant *Radiology* 49 4 oct 1947 pp 476-481
- Marqués (P.) et Pailhes (F.) Diverticule de l'œsophage et phyréno spasme *J de radiol et électrol* 28 9-10 1947
- King (B. T.) Nouvelles idées sur l'étiologie et le traitement des diverticules de l'œsophage *Surg Gynec & Obst* Chicago 85 1 1947 pp 93-97
- Jemmi (C.) Diverticule œsophagien épibronchique congénital *Oto rhino laryngol ital*, Bologna 15 5 1947 pp 294-304
- Furstenberg (A. C.) Diverticule de l'œsophage *Ann oto laryngol* 56 1947 pp 305-316
- Eichhorn (H.) Das Oesophagus Divertikel Beitrag zur Klinik Ursachenlehre und Behandlung (Mitteilung von Spätergebnissen) *Beitr klin Chir* 176 4 1947 pp 559-582
- Welt Intérêt de l'extériorisation préalable dans le traitement de certains diverticules de l'œsophage *Ann d oto-laryngol* 1947 p 704
- Ruckenstein (E.) Observations sur les origines du diverticule de Zenker *Brun s Beiter zur klin Chir* Berlin 176 3 1947 pp 333-359
- Korhonen (A.) Sur les diverticules pharyngo-œsophagiens *Duodecim* Helsinki 63 3 1947 pp 613-620
- Bauer (K. H.) Riesiges Oesophagusdivertikel *Chirurg* 17 18 1947 p 425
- Van den Wildenberg Contribution à l'étude des diverticules de pulsion pharyngo œsophagiens *La Scalpel* 100 35 1947 pp 821-827 *Ann d oto laryngol* 65 7 juil 1948
- Jackson (C. L.) Pulsion diverticulum of the hypopharynx and its surgical treatment *Ann Otol* 56 1947 p 1090
- Meda (P.) Polydiverticulose œsophago bronchique *Arch ital di anat eistol patol* Bologne an 19 vol 19 no 4 juil aout in *J de chir* 63 9 1947
- Muller (F.) Sur la pathogénie du diverticule de Zenker *Monats fur Ohrenh* Vienne 82 2 fév 1948 pp 79-83
- Shallow (T. A.) et Clerf (L. H.) Ablation des diverticules pharyngés en un temps *Surg Gynec & Obst* 86 3 mars 1948
- Shallow (T. A.) et Clerf (L. H.) Diverticulectomie pharyngée en un temps technique améliorée 186 observations *Surg Gynec & Obst* 86 3 mars 1948 pp 317-322
- Monod (Robert) Un cas de diverticule sus diaphragmatique de l'œsophage compliqué d'abcès du poulmon Traitement chirurgical Guérison du diverticule et de l'abcès pulmonaire *Soc de lar d hôp de Paris* 19 avril 1948
- Brauner (H.) Zenkersches Divertikel *Mschr Ohrenh* 82 4 avril 1948 p 187
- Forrester Wood (W. R.) Diverticule épiphyrénique de l'œsophage (avec la relation d'un cas traité chirurgicalement) *Brit J Surg* 35 140 avril 1948 pp 418-420
- Guillermi (Touzaud et Protard) Diverticule de la partie thoracique de l'œsophage *Soc d oto rhino laryng de Lyon et de la Région* 23 mai 1948 in *Lyon méd* 182 27 3 juil 1949 p 15
- Moulonguet et Hiverlet Sur deux cas de traitement chirurgical de diverticules de l'œsophage en un seul temps *Ann d oto laryngol* 65 5-6 mai juin 1948 pp 329-330
- Cocchi (U.) Diverticule par traction à l'entrée de l'œsophage et spondylose déformante de la colonne cervicale *Radiologia clin* Bale 17 4 juil 1948 pp 199-206

- Terry (R.) L'intubation de l'œsophage pour la réparation du diverticule pharyngo-œsophagien. *New-York State J Med* 48, 15 1 août 1948
- Lahey (F.) Volumineux diverticule de pulsion de l'œsophage avec complications. *Surg Gynec & Obst*, 87, 4 oct. 1948
- Adams (H. D.) Les diverticules de l'œsophage thoracique. *J Thoracic Surg*, 17 5 oct. 1948 pp 639-645
- Santy et Maillet. Diverticule épiphyrénique de l'œsophage. *Soc d'et sc sur la tub*, 13 nov 1948
- Lundgren (Nils) Epiphrenic diverticula on the basis of œsophagospasm. *Acta oto-laryn ol* 36 119, fasc 5 et 6 31 déc 1948
- Brunner (A.) Traktionsdivertikel der Speiseröhre. *Practica oto-rhino-laryngol* 10 fasc 1-2 * 1948
- Schubel (J.) Ueber Erfahrungen mit der endoskopischen Methode der Schwellendurchtrennung der Hypopharynxdivertikel. *H N O*, Berlin 1 3 août 1948 pp 123-125
- Clerc (P.) Diverticules de l'œsophage. *Actualités O R L*, 1948 Librairie Arnette Paris
- Negus (V. E.) The relation of the crico-pharyngeal sphincter to pharyngeal diverticula. *Practica oto-rhino-laryngol*, 10 fasc. 1-2 1948
- Smelt (G.) Un cas rare d'occlusion soudaine de l'œsophage. *Ned tijdschr voor geneesk* Haarlem 1, 10, 1948, pp 706-709
- Dupuis (J. P. A.) Diverticule épiphyrénique. Cure chirurgicale. *Thèse Paris* 1948
- Ahlquist (R. F.) et Kepl (M. T.) Diverticules œsophagiens. *Northwest Med* 47 7 1948 pp 493-496
- Cortesi (C.) Structure de la paroi postérieure du pharynx et de la première partie de l'œsophage en rapport avec l'existence d'un lieu de moindre résistance qui prédispose à la formation de diverticules. *Oto-rhino-laryngol ital*, Bologne 17 4 1948 pp 257-293
- Collins (E. G.) Diverticule de l'hypopharynx. Intervention. *J Laryng and Otol*, 63 1948 pp 711-712
- Bursell. Contribution au développement du diverticule pharyngo œsophagien. *Acta oto laryng* suppl 74 1948 pp 272-278
- Olivier (Cl.) Chirurgie des grands diverticules épiphyréniques de l'œsophage. *Presse méd* 17 fév 1949 no 11
- Le Roy (M. A.) Sur un cas de diverticule de l'œsophage thoracique. *Soc d' chir de Paris* (séance du 18 fév 1949)
- Poppe (J. K.) et Berg (R.) Diverticule épiphyrénique œsophagien associé au cardiospasme. *Surgery* 25 2 fév 1949, pp 231-236
- Terraoul (J.) Guernier (Y.) et Raynaud (R.) Un cas de diverticule pharyngo-œsophagien. *L O R L internat* fév 1949, no 2
- Jasonnu (D.) = Bernaglia (B.) (Bologne) Syndrome angineux chez un sujet porteur d'un gros diverticule de l'œsophage. *Minerva méd*, 40 12 10 mars 1949 pp 345-348
- Mialaret (J.) A propos des diverticules de l'œsophage thoracique et de leur traitement chirurgical. *Acad de chir* 6 avril 1949
- Monod (Robert) Trois diverticules sus phyréniques de l'œsophage ouverts dans les bronches opérés et guéris. *Acad de chir*, 6 avril 1949
- Joublin (J.) Les diverticules de l'œsophage. *Progr méd* 77, 7 10 avril 1949 pp 148-154
- Rakitova (N. I.) Un gros diverticule de la partie cervicale de l'œsophage. *Bull de chir d Grekot* Moscou 69 1 1949, pp 35-37
- Dupin (P. H. C.) Diverticules pharyngo-œsophagiens. *Thèse Paris*, 1949
- Servelle. A propos d'un diverticule de l'œsophage thoracique opéré et guéri. *Acad de chir* 27 avril 1949
- Ballivet. Diverticule épiphyrénique de l'œsophage et abcès du poulmon. Lobectomie et du verticulectomie en un temps opératoire. *Acad de chir* 11 mai 1949
- Monod (Robert) Quelques cas de diverticules de l'œsophage. *Soc de laryn l d hsp de Paris* 16 mai 1949
- Duhamel (B.) Diverticule de l'œsophage chez un nouveau né. *So de laryn ol d hsp de Paris* 16 mai 1949
- Duhamel (Bernard) Deux cas de diverticules congénitaux de l'œsophage chez des nouveau nés. *Soc de péd*, 17 mai 1949
- Herzog (P.) = Marmet (A.) A propos des diverticules de l'œsophage. Tumeur kystique épiphyrénique du médiastin postérieur de structure et d'origine vraisemblablement œsophagienne. *Acad de chir* 18 mai 1949
- Harrington (S. W.) Le traitement chirurgical des diverticules de pulsion de l'œsophage thoracique. *Ann Surg* 129 5 mai 1949

- Orator (V) Le traitement chirurgical des diverticules de l'œsophage *Wien med Wochenschr* 99 27-28, 9 juil 1949, pp 305-334
- Schlapfer (L.) Deux diverticules à la limite du pharynx et de l'œsophage chez le même malade *Schweiz med Wochenschr* Bale 79, 28 16 juillet 1949 pp 637-639
- Dohman (Lund) Opération endoscopique pour le diverticule hypopharyngé IV *Contrés internat d'oto-laryngol* Londres 18-23 juillet 1949
- Cataldo (J) Diverticulos del esofago *Rev. Asoc med argent* 83 659-660 15-30 août 1949
- McNealy et Glasmann Diverticulectomie pharyngo-œsophagienne en deux temps Indications et technique *J Internat Coll of Surgeons* 12 2 1949 pp 120-126
- Mino (R. A.) Murphy (A. J.) et Livingstone (R. G.) Diverticule œsophagien géant *Surgery*, 76 2 août 1949
- Gouze (R.) Considérations sur le traitement des diverticules de l'œsophage par l'extérèse en un temps *XLVII Congrès franç d'Oto-Rhino-Laryngol* 17 oct 1949
- Sak (L. R.) Un cas de diverticule de l'œsophage *Vjestnik O R L* Moscou III 1949 70-72
- Soulas A propos des diverticules de l'œsophage thoracique *XLVII Congrès franç d'O R L*, 28 oct 1949
- Chojnowski (J. R.) Diverticule œsophagien provoquant de l'angine de poitrine *Polski tygodnik lek* Varsovie 4 40 3 oct 1949 pp 1189-1194
- Beer (H. S.) et d'Abreu (A. L.) Les duplications de l'intestin antérieur poumon accessoire supérieur (2 cas) diverticule œsophagien épiphrenique tumeur tératoïde intra péricardique et kyste œsophagien *Brit J Surg* Londres 36 146 oct 1949
- Van den Wildenberg Des rétrécissements congénitaux et des diverticules de l'œsophage *Ann d'oto-laryngol* 65 7 In *J de chir* 65 4-5 1949
- Horowitz (S.) Pharyngo-œsophageal diverticulum *J Laryng Otol* Londres 63 10 octobre 1949
- Gonard (F.) et Badaroux. Sur la technique d'extérèse des diverticules pharyngo-œsophagiens *Afrique franç chir* no 7-8 août-octobre 1949
- Soulas Diverticules thoraciques de l'œsophage *Soc de laryngol d'hop de Paris* 21 nov 1949
- Didier (G.) et Muller (A.) Diverticule de l'hypopharynx opéré et coudure cicatricielle de l'œsophage Guérison par bougirage *Soc méd et anat chn de Lille* 30 nov 1949
- Cocchi (U.) Le diverticule de l'œsophage en radiographie *Fortschr auf dem Geb der Röntgenstr* Stuttgart 71 fasc 1 1949
- Handouza (A. S.) Quelques anomalies rares de l'œsophage *J Laryngol and Otol* London 63 4 1949 pp 217-224
- Rakitova Un volumineux diverticule de la portion inférieure de l'œsophage *Gr Surg Bull* Moscou 69 2 1949 pp 35-37
- Waldpfel (R.) Luxation du sac dans la diverticulopexie *Eye Ear Nose and Throat Monthly* 28 6 1949 pp 265-269
- Heermann (H.) Dilatation du constricteur inférieur du pharynx dans le diverticule de Zenker avec dilateur du cardia de Starck *Zeitschr für Laryngol Rhinol Otol* Stuttgart 28 5 1949 pp 262-263
- Schneider (W. E. J.) et Schemeder (P.) Le diverticule épiphrenique de l'œsophage *Zentralbl für Chir* 74 fasc 4 1949 pp 341-350 1 fig
- Bruzzone (L.) Diverticules pharyngo-œsophagiens et thérapeutique chirurgicale *Minerva chir* no 17 sept 1949 pp 482-486
- Mozolewski Diverticule sus phrénique de l'œsophage In *J de chir* 66 2 fév 1950
- Chojnowski Œsophage diverticules cause d'angor *J de chir* 66 2 fév 1950
- Hillemand (P.) Maladies de l'estomac de l'œsophage et du duodénum *Collection méd chir* Flammarion édité Paris 1950
- Soulas (M. A.) Diverticules thoraciques de l'œsophage *Ann d'oto laryngol* 67 2-3 fév - mars 1950
- Cassou (R.) et Raymond (P.) Diverticule géant de l'œsophage thoracique avec méga-œsophage *Arch de mal de l'app dig etc* 39 5 mai 1950
- Brunetti (F.) Diverticulectomia faringo esofagea con resezione e sutura trasversale in tempo unico *Arch ital di otologia* 61 1950
- Cocchi (U.) Das Oesophagusvertikel im Röntgenbild Züricher Erfahrungen an Hand von 139 Fällen (Radiographic appearances of œsophageal diverticula a study of 139 cases in Zurich) *Fortschr auf dem Gebiete der Röntgenstrahlen vereingt mit Röntgenpraxis* Stuttgart 71(1) 59-66 1949
- Dunhill (Th.) Pharyngeal diverticulum *Brit J Surgery* London 37(148) 404-415 1950
- Perrotin (J.) and Dubray Vautrin (J.) Diverticules épibronchiques de l'œsophage (Epibronchial diverticula of the esophagus) *J chir* 66(8-9) 543-556 1950

- Terry (R) L'intubation de l'œsophage pour la réparation du diverticule pharyngo œsophagien *New York State J Med* 48, 15, 1 août 1948
- Lahey (F) Volumineux diverticule de pulsion de l'œsophage avec complications *Surg Gynec & Obst*, 87, 4, oct 1948
- Adams (H D) Les diverticules de l'œsophage thoracique *J Thoracic Surg*, 17, 5 oct 1948 pp 639-645
- Santy et Maillet Diverticule épiphrénique de l'œsophage *Soc d et sc sur la tub*, 13 nov 1948
- Lundgren (Nils) Epiphrenic diverticula on the basis of œsophagospasm *Acta oto-laryngol* 36, 119, fasc 5 et 6, 31 déc 1948
- Brunner (A) Traktionsdivertikel der Speiseröhre *Practica oto-rhino-laryngol* 10 fasc 1-2 • 1948
- Schubel (J) Ueber Erfahrungen mit der endoskopischen Methode der Schwellendurchtrennung der Hypopharynxdivertikel *H N O*, Berlin 13 août 1948 pp 123-125
- Clerc (P) Diverticules de l'œsophage *Actualités O R L* 1948 Librairie Arnette Paris
- Negus (V E) The relation of the crico pharyngeal sphincter to pharyngeal diverticula *Practica oto-rhino-laryngol*, 10 fasc 1-2 1948
- Smelt (G) Un cas rare d'occlusion soudaine de l'œsophage *Ned tydschr voor geneesk* Haarlem 1, 10 1948, pp 706-709
- Dupuis (J P A) Diverticule épiphrénique Cure chirurgicale *Thèse Paris* 1948
- Ahlquist (R F) et Kepl (M F) Diverticules œsophagiens *Northwest Med* 47 7, 1948 pp 493-496
- Cortesi (C) Structure de la paroi postérieure du pharynx et de la première partie de l'œsophage, en rapport avec l'existence d'un lieu de moindre résistance qui prédispose à la formation de diverticules *Oto-rhino laryngol ital*, Bologne 17, 4 1948 pp 257-285
- Collins (E G) Diverticule de l'hypopharynx Intervention *J laryng and Otol*, 67 1948 pp 711-712
- Bursell Contribution au développement du diverticule pharyngo œsophagien *Acta oto laryng suppl* 74 1948, pp 272-278
- Olivier (Cl) Chirurgie des grands diverticules épiphréniques de l'œsophage *Presse méd*, 12 fév 1949 no 11
- Le Roy (M A) Sur un cas de diverticule de l'œsophage thoracique *Soc d chir de Paris* (séance du 18 fév 1949)
- Poppe (J K) et Berg (R) Diverticule épiphrénique œsophagien associé au cardiospasm *Surgery*, 25, 2 fév 1949 pp 231-236
- Terracol (J) Guerrier (Y) et Ravnaud (R) Un cas de diverticule pharyngo œsophagien *L O R L internat* fév 1949, no 2
- Jasonni (D) et Bertaglia (B) (Bologne) Syndrome angineux chez un sujet porteur d'un gros diverticule de l'œsophage *Minerva méd* 40, 12 10 mars 1949 pp 345-348
- Mialaret (J) A propos des diverticules de l'œsophage thoracique et de leur traitement chirurgical *Acad de chir*, 6 avril 1949
- Monod (Robert) Trois diverticules sus phréniques de l'œsophage ouverts dans les bronches opérés et guéris *Acad de chir* 6 avril 1949
- Joublin (J) Les diverticules de l'œsophage *Progr méd*, 77 7, 10 avril 1949 pp 149-154
- Rakitova (N I) Un gros diverticule de la partie cervicale de l'œsophage *Bull d chir*, de Grekov Moscou 69, 2, 1949 pp 35-37
- Dupin (P H C.) Diverticules pharyngo-œsophagiens *Thèse Paris* 1949
- Servelle A propos d'un diverticule de l'œsophage thoracique opéré et guéri *Acad de chir* 27 avril 1949
- Ballivet Diverticule épiphrénique de l'œsophage et abcès du poulmon Lobectomie et du diverticulectomie en un temps opératoire *Acad de chir* 11 mai 1949
- Monod (Robert) Quelques cas de diverticules de l'œsophage *Soc de laryngol d hsp de Paris* 16 mai 1949
- Duhamel (H) Diverticule de l'œsophage chez un nouveau né *Soc de laryngol d hsp de Paris* 16 mai 1949
- Duhamel (Bernard) Deux cas de diverticules congénitaux de l'œsophage chez des nouveau nés *Soc de pédi* 17 mai 1949
- Herzog (P) et Marmet (A) A propos des diverticules de l'œsophage Tumeur kystique épiphrénique du médiastin postérieur de structure et d'origine vraisemblablement œsophagienne *Acad de chir* 18 mai 1949
- Harrington (S W) Le traitement chirurgical des diverticules de pulsion de l'œsophage thoracique *Ann Surg* 179 5, mai 1949

- Orator (V) Le traitement chirurgical des diverticules de l'œsophage *Wien med Wochenschr* 99 27-28 9 juil 1949 pp 305-334
- Schlapfer (E.) Deux diverticules à la limite du pharynx et de l'œsophage chez le même malade *Schweiz med Wochenschr* Bale 79 28, 16 juillet 1949 pp 637-639
- Dohman (Lund) Opération endoscopique pour le diverticule hypopharyngé *IV Congrès internat d'oto-laryngol* Londres 18-23 juillet 1949
- Cataldo (J) Diverticulos del esofago *Rev Asoc med argent* 83 659-660 15-30 août 1949
- McNally et Glasmann Diverticulectomie pharyngo-œsophagienne en deux temps Indications et technique *J Internat Coll of Surgeons* 12 2 1949 pp 120-126
- Muro (R. A) Murphy (A. J) et Livingstone (R. G) Diverticule œsophagien géant. *Surgery* 26 2, août 1949
- Gourin (R.) Considérations sur le traitement des diverticules de l'œsophage par l'exérèse en un temps *LVII Congrès franç d'Oto-Rhino-Laryngol* 17 oct 1949
- Sak (L. R) Un cas de diverticule de l'œsophage *Vjesnik O R L Moscou* 11 1949 70-72
- Soulas A propos des diverticules de l'œsophage thoracique *LVII Congrès franç d'O R L* 20 oct 1949
- Chojnowski (J. R.) Diverticule œsophagien provoquant de l'angine de poitrine *Polski tygodnik lek* Varsovie 4 40 3 oct. 1949, pp 1189-1194
- Bar (H. S) et d'Abreu (A. L) Les duplications de l'intestin antérieur: poumon accessoire supérieur (2 cas) diverticule œsophagien épiphrénuque tumeur tératoïde intra péricardique et kyste œsophagien *Brit J Surg*, Londres 36 146 oct. 1949
- Van den Wildenberg Des rétrécissements congénitaux et des diverticules de l'œsophage *Ann d'oto-laryngol* 65 7 In *J de chir* 65 4-5 1949
- Horowitz (S) Pharyngo-œsophageal diverticulum *J Laryng Otol* Londres 63 10 octobre 1949
- Gonard (F) et Badaroux. Sur la technique d'exérèse des diverticules pharyngo-œsophagiens *Afrique franç chir* no 7-8 août-octobre 1949
- Soulas Diverticules thoraciques de l'œsophage *Soc de laryngol d'hôp de Paris* 21 nov 1949
- Didier (G) et Muller (A) Diverticule de l'hypopharynx opéré et suture cicatricielle de l'œsophage Guérison par bougirage *Soc méd et anat clin de Lille* 30 nov 1949
- Cocchi (U) Le diverticule de l'œsophage en radiographie *Fortschr auf dem Geb der Röntgenstr* Stuttgart 71 fasc 1 1949
- Handousa (A. S) Quelques anomalies rares de l'œsophage *J Laryngol and Otol* London 63 4 1949 pp 217-224
- Rakutova Un volumineux diverticule de la portion inférieure de l'œsophage *Gr Surg Bull* Moscou 69 2 1949 pp 35-37
- Waldapfel (R.) Luxation du sac dans la diverticulopexie *Eye Ear Nose and Throat Monthly* 28 6 1949 pp 265-269
- Heermann (H) Dilatation du constricteur inférieur du pharynx dans le diverticule de Zenker avec dilateur du cardia de Starck *Zeitschr für Laryn ol Rhinol Otol* Stuttgart 28 5 1949 pp 262-263
- Schneider (W. E. J) et Scheinader (P) Le diverticule épiphrénuque de l'œsophage *Zentralbl für Chir* 74 fasc 4 1949 pp 341-350 1 fig
- Bruzzese (L.) Diverticules pharyngo-œsophagiens et thérapeutique chirurgicale *Minerva chir* no 17 sept 1949 pp 482-486
- Mozolewski Diverticule sus phrénique de l'œsophage In *J de chir* 66 2 fév 1950
- Chojnowski Œsophage diverticules cause d'angor *J de chir* 66 2 fév 1950
- Hillemand (P) Maladies de l'estomac de l'œsophage et du duodénum *Collection méd chir* Flammarion édité Paris 1950
- Soulas (M. A) Diverticules thoraciques de l'œsophage *Ann d'oto-laryngol* 67 2-3 fév mars 1950
- Cassou (R) et Raymond (P) Diverticule géant de l'œsophage thoracique avec méga-œsophage *Arch d mal de l'app dig* etc 39 5 mai 1950
- Brunetti (F) Diverticulectomia faringo-esofagea con resezione e sutura trasversale in tempo unico *Arch ital di otologia* 61 1950
- Cocchi (U) Das Oesophagusdivertikel im Röntgenbild Züricher Erfahrungen an Hand von 139 Fällen (Radiographic appearances of oesophageal diverticula a study of 139 cases in Zurich) *Fortschr auf dem Gebiete der Röntgenstrahlen erzeugt mit Röntgenpraxis* Stuttgart 71(1) 59-66 1949
- Dunhill (Th) Pharyngeal diverticulum *Brit J Surgery* London 37(148) 404-415 1950
- Perrotin (J) and Dubray Vautrin (J) Diverticules épibronchiques de l'œsophage (Epibronchial diverticula of the esophagus) *J chir* 66(8-9) 543-556 1950

- Goodman (H I) and Parnes (I H) Epiphrenic diverticula of the esophagus *J Thoracic Surg*, 23(2) 145-159 1952
- De Bakey (M E), Heaney (J P) and Creech (O) Surgical considerations in diverticula of the esophagus *J A M A*, 1952 150/11 (1076-1082)
- Friedell (M T) and Del Beccaro (E J) Bleeding diverticula of the thoracic esophagus *Postgrad Med*, 11(3) 182-187 1952
- Perzik (S L) The transverse incision in pharyngeal pulsion diverticula *Surgery* 33(4) 544-549, 1953
- Lundskog (G E) and Stern (H) Diverticulum of the esophagus *Yale J Biol Med*, 26(4) 285-294, 1954
- Sweet (R H) Excision of diverticulum of the pharyngo-esophageal junction and lower esophagus by means of the one stage procedure A subsequent report *Ann Surg* 143(4) 433-438 1956
- Ingram (I N) and Foster (R P) Pulsion pharyngeal diverticula *J Thoracic Surg* 33,3) 287-303, 1957

Chapter 12 Abnormalities of the Veins

- Crafford et Frenckner Traitement des varices de l'œsophage *Monats fur Ohrenh und Laryngo Rhin* 1940 fasc. II
- Villaret (M), Moutier (F) Brunet (R) et Kyris (S) Etude radiologique des varices de l'œsophage *Arch d mal de l'app dig* 31 1-2 janv.-fév 1942 pp 5-27
- Blakemore (A H) et Lord (J) The technic of using vitalium tubes in establishing portacaval shunts for portal hypertension *Ann Surg*, 122, 4 oct 1945 p 476
- Steinert (G) Œsophagite ulcéreuse chronique avec observation d'un cas d'ulcère sur des varices œsophagiennes *Brit J Radiol*, mars 1946
- Trolle (Eli) et Trolle (Dure) Traitement des varices œsophagiennes par injections de produits sclérosants par voie œsophagoscopique *Acta chir Scand* 94 fasc 5, sept 1946, pp 385-397
- Higgins Jr (W H) The Œsophageal varix A report of 115 cases *Am J M Sc* 214 oct 1946 pp 436-411
- Courtney (J E) Esophageal and gastric hemorrhage *Nebraska State Med J* 31 1946 p 365
- Blakemore (A H) Indications for portacaval anastomosis Analyses of cases *Surg Gynec & Obst*, 84 15 avril 1947, pp 645-653
- Ferguson (C G) et Hackworth (L E) Hemangioma of the esophagus in Infancy *Arch Oto Laryng*, 45, mai 1947, pp 585-592
- Lathrop (F D) Injection therapy of esophageal varices *Lahey Clin Bull* 5 juil 1947 pp 143-147
- Bruetsch (H) Le problème du traitement des varices œsophagiennes *Helvet chir acta Bale* 14 4-5 oct 1947 pp 370-374
- Phemister (D B) et Humphreys (E M) Résection gastro-œsophagienne et gastrectomie totale dans le traitement des varices saignantes dans le syndrome de Banti *Ann Surg* 126 4 oct 1947 pp 397-410
- Som (M L) et Garlock (J H) New Approach to the treatment of Esophageal varices *J A M A* 135 8 nov 1947 pp 628-629
- Fabricant (N D) Varices œsophagiennes *Eye Ear, Nose and Throat Monthly* 26 12 1947 pp 650-651
- Learmonth (J R.) The problems of portal hypertension *Ann Roy Coll of Surg of England* déc. 1947 p 299
- Rowntree (L G) Zimmerman (E F) et coll Intraœsophageal venous tamponade Its use in a case of fatal hemorrhage from the esophagus *J A M A* 135 10 1947 pp 630-631
- Mersch (H J) Treatment of œsophageal varices by injection of a sclerosing solution *J A M A* 135 12, 1947 pp 754-757
- Patterson et Rouse (M O) The sclerosing therapy of esophageal varices *Gastro-Entérol* 9 4 1947 pp 391-395
- Linton (R. R.) Jones (C. M) et Volwiler Portal hypertension The treatment by splenectomy and splenorenal anastomosis with preservation of kidney *Surg Clin of North Amer* 27 1947 p 1162
- Celis (A Y) et Flores Espinosa (J) Estudio radiológico de la circulación porta a través de la vena umbilical *Rev med d Hosp Gen* 9 1947, p 289
- Fabricant (Noah D) Esophageal varices *Quarterly Review. O R L and Broncho-Esophagol* 7, 2, juin 1948

- Hare (H F) et coll Varices œsophagiennes et gastriques avec rapport d'un cas *Surg Clin North Amer* juin 1948, pp 729-732
- Davis (H H) Les varices de l'œsophage deux cas avec différents modes de traitement chirurgical *Arch Surg*, 57, 3, sept 1948 pp 391-395
- Provenzale La chirurgie des varices œsophagiennes *Arch di chir del thorace*, Florence 3 III 1948 pp 196-225
- Munoz (C E) et Mac Cormick Le traitement par injections des varices œsophagiennes dues au Schistosome de Manson Etude préliminaire *The Laryngoscope* 58 9 sept 1948 pp 992-1012
- Van Eckhoutte et W Kolff Le traitement des varices œsophagiennes saignantes par tamponnement avec un ballon de caoutchouc *Nederl tijdschr voor geneesk* Haarlem 97 47, 20 nov 1948
- Jorup (S) Varices congénitales de l'œsophage *Acta paediatr Upsal* 35 3 1948 pp 247-257
- Kelsey (M P) Robertson (H E) et Giffin (H Z) Portal obstruction in the syndrome of splenic anemia *Proc of Staff Meet Mayo Clin*, 23 1948 p 195
- Lewy (R B) Esophageal varices in Bantu's disease report of two cases in which the condition was improved with injection of sclerosing solutions *Arch Oto Laryng* 49 2 fév 1949 pp 175-183
- Tolan (J F) Esophageal varices and their treatment by injection *Laryngoscope* 59 4 avril 1949, pp 425-431
- Benhamou Dumazer et Gardel Etude radiologique des varices œsophagiennes *Soc franç d electro-radiol méd in Echos de la méd* 15 juin 1949
- Kenamore (B) et Elliot (G) The control of esophageal hemorrhage by pneumatic tamponade and thrombin *Gastroenterology* 1949 13 1 juil 1949 pp 73-76
- Hollender (L) A propos d'un cas de varices œsophagiennes traité chirurgicalement et guéri *Soc de méd de Strasbourg* 25 juin 1949 in *Strasbourg méd* 109 31-33 nov 1949
- Macbeth (R C) Injections sclérosantes des varices œsophagiennes *IV Congrès internat d oto-laryngol*, Londres 18-23 juil 1949
- Barnett (C B) et Cohen (S) The management of massive esophageal hemorrhage with tamponade and thrombin *Gastro Enterol* 1949 13 2 août 1949 pp 144-152
- Ripstein (C B) Varices œsophagiennes *Canad Med Assoc J* 61 2 août 1949 pp 141-148
- Patton (Th B) et Johnston (Ch G) Méthode de contrôle des hémorragies de varices œsophagiennes *Arch Surg* 59, 3 sept. 1949
- Weinberg (I) Observations on the occurrence of varices of the esophagus in routine autopsy material *Am J Clin Path* 19 1949 pp 554-557
- Borema (I) Le traitement chirurgical des hémorragies dues aux varices de l'œsophage dans la cirrhose hépatique et le syndrome de Bantu *Nederl Tijdschr voor geneesk* 93 50 1 déc 1949
- Linton (R R) Traitement chirurgical des varices œsophagiennes saignantes par l'anastomose porto-cave (34 observations) *Ann Intern Med Lancaster* 31 5 nov 1949 pp 794-804
- Garlock (J H) et Som (L H) Le tamponnement médiastinal comme traitement de l'hémathémèse due aux varices œsophagiennes *New York State J Med* 50 2 15 janv 1950
- Maur (M) Varices de l'œsophage *Semana med Buenos Aires* 56 5 2 fév 1950
- Carter (C) et Zamcheck (N) L'œsophagoscopie dans l'hémorragie de la partie supérieure du tractus gastro intestinal *New England J Med* 242 8 23 fév 1950
- Crile (G Jr) Transesophageal ligation of bleeding esophageal varices A preliminary report of seven cases *Arch Surg* 61(4) 654-660 1950
- Schafer (P W) and Kittle (C F) Partial esophagogastricectomy in the treatment of esophageal gastric varices *Arch Surg* 61(2) 235-243 1950
- Sengstaken (R W) and Blakemore (A H) Balloon tamponade for the control of hemorrhage from esophageal varices *Ann Surg* 131(5) 781-789 1950
- Butler (H) The veins of the esophagus *Thorax* 6(3) 276-296 1951
- Julian (O C) and Fildes (Ch E) Shunt operations for esophageal varices *Surg Clin N Amer* 31 229-258 1951
- Sheline (G F) Clark (D F) Adams (W E) and Phemister (D B) Partial gastrectomy for esophageal varices *Surg Clin N Amer* 31 213-227 1951
- Dogliotti (A M) Sulle cosiddette varici congenite dell'esofago (A proposito di un caso di telangiectasia multiple dell'esofago toracico operato di resezione) (So-called congenital esophageal varices) *G Accad med (Torino)* 114(1-12) 11-13 1951
- Dogliotti (A M) and Bruzzone (P L) Sulle cosiddette varici congenite dell'esofago (A proposito di un caso di telangiectasia multiple dell'esofago toracico operato di resezione) (So-called congenital varicosities of the esophagus Report of a case with multiple telangi-

- ectases of the thoracic œsophagus treated by resection) *Minerva med* (Torino) 1(10) 767-771 1952
- Reynolds (T B) Freedman (T) and Winsor (W) Results of the treatment of bleeding esophageal varices with balloon tamponade *Am J Med Sci*, 224(5) 500-506 1952
- Wagenknecht (Th W) Noble (J F) and Baronofsky (I D) Nature of bleeding in esophageal varices *Surgery* 33(6) 869-874, 1953
- Robert (F) and Hoffmann (T) Zum Nachweis der Ösophagusvarizen und ihrer klinischen Bedeutung beim portalen Hochdruck (Detection of esophageal phlebectasias and their clinical importance in portal hypertension) *Fortschr Röntgenstr* 79(1) 51-70 1953
- Linton (R R) and Warren (R) The emergency treatment of massive bleeding from esophageal varices by transesophageal suture of these vessels at the time of acute hemorrhage *Surgery*, 33(2) 243-255, 1953
- Chiles (N H), Baggenstoss (A H) Butt (H R) and Olsen (A M) Esophageal varices comparative incidence of ulceration and spontaneous rupture as a cause of fatal hemorrhage *Gastroenterology*, 25(4) 565-573 1953
- Linton (R R) The emergency and definitive treatment of bleeding esophageal varices *Gastroenterology*, 14(1) 1-9 1953
- Crile (G Jr) Treatment of esophageal varices by transesophageal obliteration *Surg Gynec & Obst* 96(5) 573-576 1953
- Palmer (E D) Sbrovov (V M) and Jahnke (F J Jr) Effects of surgical treatment of esophageal varices on the portal venous pressure and hepatic function preliminary observations *Gastroenterology*, 24(1) 10-19, 1953
- Bennett (H D) Lorentzen (C) and Baker (L A) Transient esophageal varices in hepatic cirrhosis *Arch Intern Med* 92(4) 507-522 1953
- Brick (I B) and Palmer (E D) Incidence and diagnosis of esophageal varices in cirrhosis of the liver an esophagoscopic study *Gastroenterology*, 25(3) 378-384 1953
- Morton (J H) and Whelan (T J Jr) Esophageal varices without portal hypertension. Case report *Surgery*, 36(6) 1138-1143 1954
- Palmer (E D) Emergency treatment of bleeding esophageal varices (Treatment by transesophagoscopic sclerosing injection plus pneumatic tamponade) *Arch Otolaryng* 59(5) 536-542 1954
- Palmer (E D) and Brick (I B) Esophageal varices in noncirrhotic patients Esophagoscopic study *Am J Med* 17(5) 641-644 1954
- Cooley (D A) and DeBakey (M E) Subtotal esophagectomy for bleeding esophageal varices *Arch Surg*, 68(6) 854-871 1954
- Hallenbeck (G A) and Shocket (E) Treatment of bleeding esophageal varices after splenectomy *Arch Surg*, 71(4) 581-587, 1955
- Linton (R R) and Ellis (D S) Emergency and definitive treatment of bleeding esophageal varices *JAMA*, 160(12) 1017-1023, 1956
- McDermott (W V Jr) Wareham (J) and Ruddell (A G) Bleeding esophageal varices A study of the cause of the associated hepatic coma *Ann Surg* 144,3) 318-335 1956

Chapter 13 Acute and Chronic Esophagitis

ACUTE ESOPHAGITIS, ETC

- Guarnaccia Micro-diverticulose kystique suppurée de l'œsophage *L O R L ital* 10 5 sept. 1940
- Hæberlin Œsophagite exfoliatrice *Schweiz med Woch*, 5 aout 1944 p 827
- Castellanos (A) and Codinach (A) Œsophagite chez un enfant. Considérations diagnostiques *Arch de méd inf* 14 3, 1945 pp 175-197
- Welin (S) Contribution au diagnostic radiologique de l'œsophagite limitée *Acta radiol* 27 3-4 1946 pp 461-471
- Bergquist Contribution à l'étude de l'œsophagite limitée *Acta oto-laryngol* 34 1-3 1946 pp 256-278
- Wolcott (C. C.) Periesophagitis *Laryngoscope* 57, 1 janv 1947 pp 45-56
- Steiner (G) Œsophagite ulcéreuse chronique dans un cas d'ulcère œsophagien avec varice *Ann d oto-laryngol* 64, 10 oct 1947
- Malossini (E) Œsophagite ulcéreuse simple *Il Valalta* 23 1-2 1947 pp 1-11
- Olsen (A) Esophagitis *Surg, Gynec and Obst* 86 3 mars 1948
- Belinoff (S) A propos de l'œsophagite catarrhale *Monats fur Ohren* no 5-6 mai juin 1948
- Maspetoul et Hacker Tétanos viscéral subaigu à point de départ œsophagien (œsophagite segmentaire latente) *Soc de laryngol d hôp de Paris in Ann d'oto-laryngol* déc. 1948

- Cattan (R.) Frumusan (P.) Brulé (G.) et Cousin. Un cas d'œsophagite œdémateuse rebelle. Guérison par le 3277 R. P. *Soc nat franç de gastro entérol*, 14 mars 1949 in *Presse méd* no 30 7 mai 1949
- Willcox. Moule œsophagien simple. *The Lancet*, 7 III 1949 pp 417-418
- Franklin (R.) et Taylor (S.) Oesophagite granulomateuse non spécifique régionale. *J Thoracic Surg* 19, 2 fév 1950
- Nemours Auguste. Réflexions sur le reflux cardio œsophagien. *Soc nat franç de gastro entérol* 15 mai 1950

- Ferguson (D. J.) et al. Studies on experimental esophagitis. *Surgery* 28 1022-1042, 1950
- Franklin (R. H.) and Taylor (S.) Nonspecific granulomatous (regional) esophagitis. *J Thoracic Surg* 19(2) 292-297 1950
- Mason (L. B.) and Ausband (J. R.) Benign stenosing esophagitis associated with vomiting and intubation. *Surgery* 32(1) 10-16 1952

PLUMMER VINSON SYNDROME

- Vinson (P. P.) Hysterical Dysphagia. *Minnesota Med* 5 1922 p 107
- Hurst. Hysterical Dysphagia. *Guy's Hosp Rep*, 76 1926 p 426. *Brit M J*, 1928
- Mersch et Connor. The dysphagia. *Arch Oto Laryng* 4 1926 p 112
- Hill (W.) The Syndrome Plummer Vinson. *Brit M J* 1 1928 p 467
- Cameron. The Plummer Vinson Syndrome. *J Laryng and Otol* 43 1928 p 219. *J Laryng and Otol* 44 1929 p 168
- Evans (G.) The Plummer Vinson Syndrome. *Practitioner* 124 1930 p 317
- Johnson. Hysterical dysphagia. *Proc Roy Soc Med (Sect Laryng)* 24 1931 p 1206
- Witts. The Plummer Vinson Syndrome. *Guy's Hosp Rep* 81 1931 p 193
- Suzmann. Syndrome of Anemia Glossitis and Dysphagia. *Arch Int Med* 51 1933 p 1
- Hallen. The syndrome of dysphagia with anemia. *Acta med Scand Suppl* XC 1937 p 398
- Thiele et Kuehl. Ueber die Essentielle Hypochrome Anämie. *Klin Wschr* 2 1938 p 1191
- Beltrametti et Balestrieri. Due casi di anemia ipocromica essenziale con sindrome di Plummer Vinson. *Gazz degli Osp e delle Cl* 45 1939 p 999
- Ferretti (S.) Su due casi di sindrome Anemia Disfagia. *L Ateneo parmense* 1939
- Waldenström et Kjellberg. The Röntgenological diagnosis of sideropenic dysphagia (Plummer Vinson syndrome). *Acta radiol* 20 1939 p 618
- David P. Cordray. Syndrome de Plummer Vinson. *Ann Otol Rhinol and Laryngol* 49 1 mars 1940
- Gerlings (P. G.) Disorders of the mouth of the esophagus in the syndrome of Plummer Vinson. *J Laryngol and Otol* 55 3 mars 1940 p 149
- Terracol (J.) et Lamarque (P.) Le syndrome de Plummer Vinson. *Montpellier méd* janv 1941
- Meulengracht et Bichel. Riboflavina Avitaminosi und das Plummer Vinson Syndrome. *Klin Wchschr* 1941 p 831
- Meyer. Avitaminosi riboflavonica e sindrome di Plummer Vinson. *Klin Wchschr* 2 1942 p 39
- Massimello. La sindrome di Plummer Vinson. *Minerva Med* 34 1943 p 177
- Pietrantonio et Traverso. Syndrome di Plummer Vinson e anemia ipocromica essenziale. *Riv clin med* 28 1943 p 2
- Weder. Syndrome di Plummer Vinson. *Schweiz med Wochen* 73 1944 p 45
- Moulounguet. Un cas de syndrome de Plummer Vinson (dysphagie par sideropenie). *Soc de laryngol d hop de Paris in Ann d oto laryngol* oct déc 1945
- Baumgartel. Spasme de l'œsophage et syndrome de Plummer Vinson. *Wien klin Wchschr* 27 déc 1946 p 780
- Dubois Fernier. La sindrome di Plummer Vinson. *Praxis* 51-52 1946 p 886
- Sisto (P.) La sindrome di Plummer Vinson. *Gazz San* 49 1946
- Vannotti (A.) Syndrome di Plummer Vinson alterazioni epiteliali disturbi digestivi acclorid. *Schweiz med Woch* 4 12 1946 pp 309-310
- Lederer (J.) et Renner (M.) Le syndrome de Plummer Vinson et son traitement par la thérapeutique martiale parentérale. *Acta gastro enterol belgica* Bruxelles 10 2 fév 1947
- Ran (L.) Syndrome de Paterson Plummer Vinson dans un cas d'ictère familial. *Proc Roy Soc Med* Londres 40 6 avril 1947
- Thomas (M. A.) Membranes et bandes de constriction de la partie supérieure de l'œsophage (dysphagie sideropenique). *Am J Röntg* 57 2 1947 pp 213-219
- Pieranceli. A propos du syndrome de Plummer Vinson. *Boll d malat d'orecchio* Florence 65 7-8 1947 pp 137-145

- Huizinga Eelco Chlorosis tarda met dysphagie en carcinoom *Ned tydschr voor geneesk*, no 1, janv 1948
- Videbech (G) Cinq cas de syndrome de Plummer Vinson *Monats fur Ohrenh* In *Rev de laryngol*, nos 5 et 6, mai juin 1948
- Cojazzi (L) Sulla patogenesi della sindrome di Plummer Vinson e di affini alterazioni atrofiche della mucosa orale e faringea *Atti del XVIII Congr della Soc ital di O R-L*, 1948
- Comu (G) Pathogénie de l'anémie hypochrome essentielle et du syndrome de Plummer Vinson *Marginal oto laryngol* Florence, 6, 1, 1948, pp 76-96
- McGee et Goodwin The syndrome of dysphagia and anemia *Ann Int Med* 11, 2 1948 p 1438
- Gernez (A) *Diplome de radiologie* Faculté de Médecine Paris, 1948
- Pustorino (D) Anemia ipocromica Sindrome di Plummer Vinson *Policl infant*, 184 1948
- Secco (C) et Cavallari (A) Considerazioni sopra un caso di malattia di Plummer Vinson *Minerva med* 48, 1948, p 631
- Wahlquist (H F) Syndrome de Plummer Vinson Rapport d'un cas *An espagn I d'odontostomatol*, Madrid, 7 9 1948, pp 833-836
- Thornval (A) Spasms in the upper region of the oesophagus partly with and partly without Plummer Vinson syndrome *Acta oto laryngol*, Helsinki, suppl 1948 67, pp 131-133
- Savilahti (M) On the Pathologic Anatomy of the Plummer Vinson Syndrome *Acta med Scand* 135 1 1948
- Szenes (H) Zwei Falle von Plummer-Vinson Syndrome *Monats fur Ohrenheilk*, Wien 83 1, janvier 1949 pp 37-39
- Szenes (H) Sur le syndrome de Plummer-Vinson *Monats fur Ohrenheilk*, avril juin 1949, Heft 4-5-6
- Gernez (A) L'intérêt du syndrome de Plummer Vinson en cancérologie *Paris méd* 39 18 14 mai 1949, pp 202-208
- Ferrando Botey (J M) Considérations sur le syndrome de Plummer Vinson *Ann d'oto-laryngol* 66, 1-2 1949
- Léger (L), Gernez et Auvert Les dysphagies sidéropives (syndrome de Plummer Vinson) *Soc nat franç de gastro entérol* in *Presse méd* 13 aout 1949
- Gernez (A) Dysphagie sidéropénique et membranes œsophagiennes *Presse méd* no 26 20 avril 1949
- Cojazzi (L) et Bonino (R) Sulla patogenesi e sulla clinica della sindrome di Plummer Vinson *Arch ital di otol*, 60, 6 nov déc. 1949
- Bonorino Udaondo Syndrome de Plummer Vinson *Soc nat franç de gastro entérol*, 9 janv 1950, in *Presse méd* no 12 26 fév 1950 p 204
- Bonorino Udaondo c, d Alotto (V) et Resano (H) Syndrome de Plummer Vinson *Arch mal app dig* etc, 39, 4, avril 1950
- Brulé (G) Le syndrome de Plummer Vinson *Gaz méd de France*, 57, 10 mai 1950
- Dury Un cas de syndrome de Plummer Vinson *Bull Soc O-R L Lyon* fasc 3, mai et juin 1950
- Lanzara (A) Osservazioni cliniche di esofagite peptica dopo interventi sul cardias II Studio clinico-radiologico del reflusso gastro esofageo e digiuno-esofageo postoperatorio (I Observations on peptic esophagitis after cardia resection II Clinical studies on post operative reflux esophagitis) *Arch chir* (Torace) 7 41-90 1951
- Bingham (J A W) and Logan (J S) The nature and treatment of Plummer Vinson dysphagia *Brit Med J* 1953 no 4837 (pp 650-652)
- Juddles (A) Plummer Vinson syndrome Report of case treated surgically *Delaware St Med J*, 26(10) 264-267, 1954

Chapter 14 Esophageal Abscess, Phlegmonous Esophagitis, and Periesophagitis

- Neuhof Acute infections of the mediastinum with special reference to mediastinal suppuration *J Thoracic Surg*, 6 1936 p 184
- Adams Acute suppurative mediastinitis *J Thoracic Surg*, 15 1939, p 336
- Lezius Therapy of acute suppurative mediastinitis *Arch fur klin Chir* 196 1939 p 616
- Pallestrini Inflammation de l'œsophage et du médiastin par corps étranger dans l'œsophage Traitement chir *Acad de méd* Genova 54 1939 p 228
- Ruis (M) Suppuration du médiastin postérieur, nouvelle méthode de drainage Un cas de guérison *Ann d'oto-rhino-laryngol* Uruguay 9, 1939 p 186
- Donald Médiastinite antérieure *J de chir*, 56 1940

- Marschik (H) History of mediastinotomy *Wien klin Wochenschr* 53, 1940 p 360
- Burnett Recognition and Management of Mediastinitis *Am J Surg* 54 1941
- Löbbel La médiastinite par corps étranger *Deutsche med Wochenschr* 68 21 1942
- Szecsey Complications médiastinales à la suite de maladies de l'œsophage *Oto rhino laryngol ital* 13 1 janv. fév. 1943
- Santy (P) et Bérard (M) Phlegmon gangreneux du médiastin d'origine œsophagienne Médiastinite Pénicilline *in situ* Guérison *Soc d chir de Lyon* séance du 21 juin 1945 p 101
- Foco (G) Sur un cas d'abcès rétro pharyngo œsophagien *Minerva med* 36 48 1945 pp 235-237
- Grez Trois cas de médiastinites par perforation de l'œsophage Pénicilline Guérison *Bolet della Soc de chir de Chili* Santiago 23 2 1945
- Ramos et De Almeida (J M) Penicillin therapy of acute mediastinitis *Rev brasil med* 1945 p 31
- Sutton Abcès dû à une perforation par morceaux de verre *Canad M A J* 52 1945 p 82
- Perotin Lemaire et Stœcklin Technique de la méthode de Philips et Van Slycke *Presse méd* 9 fév 1946
- Ballivet Abcès cervical rétro-œsophagien par corps étranger *Bull et mém Acad de chir* no 29 1946 p 487
- Gurd (F B) Le diagnostic des affections médiastinales *Surg Clin North Amer* 26 2 1946
- Gimbrère (A F) Abcès rétro œsophagien *Nederl tijdschr voor geneesk* 90 1946
- Davis (J E) Dissection traumatique de l'œsophage Médiastinite avec guérison *Arch Oto Laryngol* 237 1946
- Latraverse De l'abcès de l'épiglotte à l'œsophagite phlegmoneuse *L'Union méd du Canada* Montréal 69 10 oct *In J de chir* 62 7-8 1946
- Gardères et Derron Corps étranger de l'œsophage inextirpable par voies naturelles Infection gangréneuse Œsophagostomie par les voies naturelles sans sutures profondes Sutures cutanées Pénicilliothérapie Guérison *Acad de chir* 1946
- Mosher (H P) Infections péri œsophagiennes dans les maladies générales *Ann oto laryngol* 13 9-10 1946 p 476
- Mayoux et Martin Médiastinite septique guérie par l'association sulfamide pénicilline *Lyon méd* 1946
- Grez (A) Deux cas de médiastinite avec perforation de l'œsophage Traitement à la pénicilline Guérison *Rev argent de broncho esofagol* 1 1946 pp 70-76
- Ravalet Bourret Guinet et Garde Ostéo arthrite infectieuse du rachis cervical consécutive à la blessure du pharynx par un corps étranger accidentellement dégluti *Lyon méd* 78 34 1946
- Moatti Pyo-pneumothorax secondaire à un bougirage de l'œsophage *Tunis méd* 35 2 mars avril 1947
- Belkina (N P) To the question on the surgical treatment of peri œsophagus *Vestnik oto rino laryngol* no 3 1947 pp 61-64
- Hunermann Médiastinite après traitement des sténoses cicatricielles de l'œsophage et autres interventions de l'œsophage *Arztliche Forsch Bad Worish* 1 6-7 1947 pp 138-141
- Wolcott (C C) Péri œsophagite *The Laryngoscope* 57 5 1947 pp 45-56
- Lyman (E H) Un cas de corps étranger de l'œsophage avec médiastinite et autres complications *The Laryngoscope* 57 6 1947 pp 400-413
- Pagano Les complications médiastinales de suppurations péri pharyngées *Bol d mal d orecchio d gola d naso* Florence 65 1-2 1947 pp 31-32
- Ferreri (G) Abcès rétro cricoidien dans une médiastinite purulente *Bol d mal d Orecchio d Gola d Naso* Florence 65 3-4 1947 pp 63-64
- Likely (S W) Complications médiastinales en rapport avec l'œsophagoscopie *Ann Oto Laryngol* 64 7 1947 p 392
- Hammond (A P) Médiastinotomie cervicale d'urgence *Ann d oto laryngol* 1947 p 395
- Didier (G) et Clerc (A) Les phlegmons médiastinaux péri œsophagiens *J des Sc méd de Lille* 1 février 1948
- Homb (A) Le traitement de la médiastinite par perforation de l'œsophage *Acta chir Scand* Stockholm 97 fasc 3 21 dec 1948
- Clark (J V) Un cas d'abcès sous muqueux de l'œsophage *J Laryngol and Otol* London 62 7 1948 pp 461-464
- De Weese Diagnostic et traitement de la médiastinite aigue suppurée Rapport de six cas *Trans Pacific Coast Oto ophthalmol Soc* 29 1948 pp 183-196
- Schumann (H) Emploi d'un tampon spongieux dans le traitement de la médiastinite et de la

- fistulisation pleurale, avec alimentation par les voies naturelles *H N O* Berlin 1, 4, 1948, p 179
- Nys (P H J) Abscès traumatique de l'œsophage *Ann belges de méd milit* Liège 101 7-8 1948 pp 123-125
- Weinhold Médiastinal abcesse *H N O, Beih z Z Hals Heilk u s w* 1, 1948 p 134
- Jemern et Neuhof Demonstration of cervico mediastinal continuity with comments on extra pleural extension of mediastinal abcess *J Thoracic Surg*, 9 1948 p 1
- Joussemet (R) Médiastinites aiguës postérieures par plaies endo œsophagiennes *J de ch* 1948
- Andersen (I) et Poulsen (T) Le traitement de la médiastinite aiguë et de la perforation œsophagienne *Nordisk med*, 41 21 27 mai 1949 pp 943-947
- Duhamel (B) et Autier (Ch) Médiastinite aiguë suppurée après perforation traumatique de l'œsophage chez un enfant de 28 mois Intervention Guérison *Soc de péd*, 20 déc 1949
- Theilkaas (E) et Ludin (M) Un cas de perforation de l'œsophage avec médiastinite consécutive *Radiologica clinica*, Bale janvier 1950 pp 57-61
- Guisez *Diagnostic et traitement des rétrécissements de l'œsophage et de la trachée* Masson et C^e édit
- Ballivet and Truffert Abscès cervical rétro œsophagien par corps étranger Pénicilline Cervicotomie Aspiration (Retro esophageal cervical abscess due to foreign body Penicillin Cervicotomy Aspiration) *Sem d Hop de Paris* 23(1) 17 1947

Chapter 15 Nonspecific Ulcerations

- Feldmann (M) Deux cas d'ulcère peptique de l'œsophage inférieur associé à une hernie gastrique *Am J Med Sc*, août 1939 p 165
- Chamberlin (D T) Peptic ulcer of the œsophagus *Am J Digest Dis* 5, 1939 p 725
- Dick (R C S) et Hurst Chronic peptic ulcer of the œsophagus and its association with congenitally short œsophagus and diaphragmatic hernia *Quart J Med*, 11, 1942 pp 105-120
- Lamblin Aubin et Debray Maladie ulcéreuse œsophagienne et probablement duodénale gastrite congestive récidivante, lithase vésiculaire *Soc de gastro-entérol* Paris 9 oct 1943 pp 10-11
- Allison (P R) Peptic ulcer of the œsophagus *J Thoracic Surg* 15 1946 pp 308-317
- Preisler Peptic œsophageal ulcer *Lancet*, no 6397 250 1946 pp 497-498
- Steiner (G) Ulcère œsophagien sur de varices œsophagiennes *Brit J Radiol*, avril 1946
- Morton (D R) et Brunschwig (A) Peptic ulcer of œsophagus *Gastro Entérol*, 7 3 1946 pp 314-319
- Diaz (F) et Riera (M) L'ulcère peptique de l'œsophage *Rev med de Chile* Santiago 73, 12 In *J de chir* 67 7-8, 1946
- Seuders Sur l'ulcère peptique de l'œsophage *Arch ital di otol*, 57 fasc 4 1946 p 149
- Brandsma (A G) Perforation en deux temps d'un ulcère peptique de l'œsophage *Nederl tydschr voor gen* 90 27 5 juil 1947, pp 1794-1799
- Finocchi (G) Contribution à l'étude du phénomène de Schwarzmann dans une localisation œsophagienne *Il Valsala* 23, 9-10 1947 pp 148-155
- Allison (P R) Ulcère peptique de l'œsophage *J Thoracic Surg* 3 1 janv 1948 pp 20-42
- Castleton (K B) et Dolowitz (D A) Ulcère peptique de l'œsophage avec hémorragie grave traitée par l'oxycel *Gastro Entérol* 10 5 mai 1948 pp 797-801
- Hartz (I H) et Van der Sar (A) Ulcère aigu œsophagien avec inclusion intranucléaire *Gastro Entérol* 11 3 sept 1948 pp 337-341
- Benedict (E) et Sweet (R) Ulcère béniin de l'œsophage *Gastro Entérol* 10 5 nov 1948
- Benasi (E) L'ulcère peptica dell'esofago *Arch ital mal app dig* 14 5-6 1948 pp 363-385
- De Amesti et Chechilutzky (L) Ulcère peptique avec sténose de l'œsophage Œsophago-gastrectomie *Rev med de Chile*, Santiago 76 4 1948 pp 232-235
- Naulleau (J) et Sarazin Ulcère peptique de l'œsophage thoracique *Soc franç d'El cirrual* 1 *méd* (Fihale de l'Ouest) Angers, 23 janv 1949 in *J de radiol* 30 11-12 1949
- Berdal (P) Œsophagite peptique Ulcère peptique de l'œsophage *Nord med* 4 19 avr 1949
- Thomsen (G) L'ulcère peptique de l'œsophage *Acta radiol* Stockholm 37 2-3, 30 sept 1949 pp 193-209
- Gerlings Pathogénie et thérapie de l'ulcère peptique de l'œsophage *Congrès franç d'oto-rhino-laryngol* 20 oct 1949 in *Bronchosc*, *Oes phar osc* et *Gastrosc* 1949

- Jentzer Ulcère de la partie inférieure de l'œsophage avec diverticule sus et sous diaphragma-
tique *Rev. méd. Suisse romande* nov 1949 p 78
- Churchill (E D) Ulcer of esophagus *New England J Med* 240 23 1949 p 927
- Wangensteen et Leven (L) Résection gastrique pour œsophagite et rétrécissement d'origine
peptique *Surg. Gynec. & Obst.* 88 5 1949 pp 560-570
- Berlin Ulcère de l'œsophage et hyperplasie thyroïdienne *J de chir.* 66 2 fév 1950
- Erskine (F A) Mason (J H) et Mac Dane (H) Perforations d'ulcères de l'œsophage au
cours de la poliomyélite bulbaire *Am J Med* 8 2 fév 1950 pp 239-245
- Conette (P) Ulcère peptique de l'œsophage *Thèse Paris* 1950 p 186
- Udaondo (C B) et d'Alotto (V) Œsophage court et ulcères du cardia *Arch. d. mal. de
l'app. dig.* 39, 9-10, sept-oct 1950
- Ferguson (D J) Sanchez Palomera (E) Sako (Y) Clatworthy (H W Jr) Toon (R W)
and Wangenstein (O H) Studies on experimental esophagitis *Surgery* 28(6) 1022-
1039 1950
- Barrett (N R) Chronic peptic ulcer of the œsophagus and œsophagitis *Brit J Surg*
38(150) 175-182 1950
- Allison (P R) Refluxœsophagitis sliding hiatal hernia and the anatomy of repair *Surg
Gynec. & Obst.* 92(4) 419-431 1951
- Bosher (L H Jr) and Taylor (F H) Heterotopic gastric mucosa in the esophagus with
ulceration and stricture formation *J Thoracic Surg* 21(3) 306-312 1951
- Lortat Jacob (J L) Les maladies peptiques de l'œsophage (Peptic diseases of the esophagus)
J Int. Chir. 11(2) 152-175 1951
- Cross (F S) and Wangenstein (O H) Role of bile and pancreatic juice in production of
esophageal erosions and anemia *Proc. Soc. Exp. Biol. (N Y)* 77(4) 862-866 1951
- Lund (E) Ulcerative esophagitis complicated by severe hemorrhage *Acta otolaryng. (Stock-
holm)* 43(Suppl. 109) 99-101 1953
- Wolf (B S) Som (M) and Marshak (R H) Short esophagus with esophagogastric or marginal
ulceration *Radiology* 61(4) 473-495 1953
- Allison (P R) and Johnstone (A S) The œsophagus lined with gastric mucous membrane
Thorax 8(2) 87-101 1953
- Carver (G M Jr) and Sealy (W C) Peptic esophagitis *Arch. Surg.* 68(3) 286-295 1954
- Benedict (E B) and Gillespie (J E O N) Esophageal stenosis caused by peptic esophagitis
or ulceration *New England J Med* 250(15) 642-651 1954
- Sweet (R H) Robbins (L L) Gephart (Th) and Wilkins (E W Jr) The surgical treat-
ment of peptic ulceration and stricture of the lower esophagus *Ann Surg* 139(3) 30(3)
258-268 1954
- Counaud (C) Maladie peptique de l'œsophage chez l'adulte Etat actuel de la question
(Peptic ulcer of the esophagus in the adult Present state of the problem) *J chir. (Paris)*
71(5) 408-427 1955
- Cross (F S) and Kay (E B) The etiology and treatment of peptic esophagitis *Ann Surg*
143(3) 360-368 1956
- Som (M L) and Wolf (B S) Peptic ulcer of the esophagus and esophagitis in gastric lined
esophagus *New England J Med* 162(7) 641-644 1957
- Winkelstein (A) Peptic esophagitis with duodenal ulcer *Am J Surg* 93(2) 234-237 1957

Chapter 16. Specific Infections

TUBERCULOSIS

- Kragh (J) Tuberculous diverticula of the esophagus *Acta oto laryngol.* 4 1922-1923 pp
49-62
- Parvis (A) Contribution à l'étude de la tuberculose de l'œsophage *Arch. ital. mal. trach.
ecc.* 7 1939 pp 231-242
- Clerf (L H) Sténose par abcès tuberculeux péri œsophagien. XVIII Réunion de l'Assoc. de
Broncho Œsophagol. New York City 5 juin 1940
- Elizalde (P) et Latuada (R) La tuberculose ulcéreuse de l'œsophage *Arch. de la Soc. argent.
de anatomia normal y path.* Buenos Aires 2 1 5 avril août 1940
- Pretzman (J A) Fall von endoskopisch diagnostizierte Œsophagus-Tuberkulose *Nord. méd.*
1942
- Aubin (V M) Un cas de tuberculose hypertrophique de l'œsophage avec dégénérescence
cancéreuse *Soc. de laryngol. d'hygiène de l'urine séance du 21 juin 1943*
- Meda (P) Contribution à l'étude de la tuberculose œsophagienne *Arch. ital. d'otol. rin. e
laryng. fasc.* 4, 1943

- Zoltan Die Tuberculose der Speiseröhre *Acta oto laryngol* 31 1943 pp 32-39
- Hawes (L. E.) Röntgenologic changes in Esophagus in Tuberculous Mediastinitis *Am J Röntgenol and Rad Therapy* 11 51, 1944 p 575
- Hamberger (C. A.) De la tuberculose de l'œsophage *Acta oto-laryngol*, Stockholm 33 4-5 1945 pp 281-288
- Villata (L.) Tuberculose miliaire de l'œsophage II *Valsalva* Rome 73, 7-8 juil aout 1947, pp 85-94
- Ludin (N.) L'aspect radiologique de la tuberculose de l'œsophage *Schweiz. Zeitschr. für Tuberk.* Bale 4 4, 1947 pp 267-272
- Cistaro (A.) Ricerche istopatologiche sistematiche sull'esofago di soggetti tubercolotici di particolari strutture arteriose della sottomucosa esofago *Oto-rino-lar ital* 16, 1948 p 325
- Jeanne (E.) Considérations nouvelles sur la tuberculose de l'œsophage traité par l'œsophagectomie *Thèse Paris*, 1948
- Majeranowski (J. F.) Observations histologiques sur deux cas de tuberculose de l'œsophage *Archiv Path* 45 608-615, 1948
- De Fazio Fistule œsophago-épigastrique tuberculeuse *Riforma med* 63 7, 19 fév 1949, pp 145-150
- Petrovski Tuberculose de l'œsophage *Problème de la tub.* Moscou, 2 1949, pp 47-69
- Lawson (J. F.) Fistule tuberculeuse œsophago-cutanée traitée par streptomycine et gastrostomie *Am Rev Tubercul* juin 1949
- Heimendinger (H.) Frühling (L.) et Klotz (G.) Tuberculose de l'œsophage développée sur une sténose cicatricielle préexistante et traitée par la streptomycine *Rev. de laryng. otol. rhin.* Bordeaux a 70 9-10 sept-oct 1949
- Audouin (J.) et Poulain (J.) Tuberculose sténosante de l'œsophage d'apparence primitive guérie par œsophagectomie *Soc. nat. franç. de gastro-entérol*, 14 nov 1949 in *Pres. méd.*, 17 déc 1949
- Guillaume (Edouard) Contribution à l'étude clinique de la tuberculose de l'œsophage *Thèse Paris* 1949
- Hoidt (G.) Un cas de tuberculose œsophagienne *Acta oto laryng* Stockholm, 30 p 570
- In *Ann. d'oto-rhino-laryngol* 13 5-6
- Zavalla (A. T.) Torres (M. L.) and Dimayuga (R.) Tuberculosis of the esophagus and stomach. Report of a case *Philippine J. Surgery* 5(3) 111-114 1950
- Audouin (J.) and Poulain (J.) Tuberculose sténosante de l'œsophage d'apparence primitive guérie par œsophagectomie (Stenosing tuberculosis of the esophagus apparently primary cured by esophagectomy) *Arch. mal. appar. dig.* 39(2) 231-236 1950
- Resano (H. J.) Carlos Rey (J.) Malenchini (M.) and De Diego (R.) Estenosis del esófago por tuberculosis (Stenosis of the esophagus caused by tuberculosis) *Medicina* (B Aires) 13(3) 182-190, 1953
- SYPHILIS
- Gurjot Etude sur l'anatomie pathologique de la syphilis œsophagienne *Ann. d'oto-laryngologie* 1931
- Fontana (D.) Stenosi luetica esofagea a sintomatologia simulante una fistola esofago-tracheale *Ann. laryng.* fasc. 1 1938
- Faroy (G.) et Paillis Sténose cardio-œsophagienne d'origine syphilitique probable avec association d'atrophie gastrique totale *Arch. d. mal. de l'app. dig. et des mal. de la nutrit.* 31 1942 p 265
- Malfaut (G.) La sifilide dell'esofago *Boll. d. malat. d. orecchio gola naso etc.* Firenze nov. 1942
- Ségal (M.) Sténose syphilitique de l'œsophage *South African Med. J.* 27 juil 1946
- Creyx Laumonier (P.) Lévy (J.) Texier (L.) et David Chausse (J.) Diagnostic étologique litigieux d'une sténose œsophagienne (syphilis probable) *Soc. de m. d. et chir. de Bordeaux* 21 mai 1948
- Meyer Bothling (H. J.) Ueber die luetische Speiseröhren-Entzündung (I ues II) *Zeitschr. Laryng. u. s. w.* 1 1948 pp 71-72
- Auguste (C.) Breton (A.) and Paris (J.) Fistule trachéo-œsophagienne chez une syphilitique (Tracheo-esophageal fistula in a syphilitic) *Arch. mal. appar. dig.* 38 9-10 1007-1012 1949
- Hudson (T. B.) and Head (J. R.) Syphilis of the esophagus *J. Thoracic Surg.* 10(1) 216-220 1950

DIPHTHERIA

- Rolleston (R. D.) Diphthérie de l'œsophage *Brit J Child Dis* 9 janv 1912 pp 12-16
- Stupka Die Diphtherie der Speiseröhre und ihre Folgezustände (Narbenstenosen) *Zeitschr f Chir*, 170 1922 pp 1-52
- Gonin (W. H.) Kasuistischer Beitrag zur Kenntnis der echten Diphtherie des Oesophagus und des Magens *Schweiz med Wschr* 53 1923 pp 625-626
- Spitz Die echte Diphtherie der Speiseröhre und des Magens *Zeitschr f Laryngol u s w* 13, 1925 pp 136-174
- Peut (R.) A propos d'un cas fatal de diphthérie généralisée *Bull et mém de la Soc med de Paris* 1926, pp 391-393
- Lemmel Diphthérie de l'œsophage *Arch f Verd* juin 1928, anal in *Paris méd* 8 juin 1929
- Guminski Un cas d'oidium albicans avec diphthérie de l'œsophage *Polski Przegl Otol*, 6 1929 pp 45-52
- Collet (F. J.) Hémoplégie pharyngo-laryngée et paralysie œsophagienne post-diphthérique *Soc de laryngol hôp Paris* 16 mars 1931
- Brandt (G.) Oesophagus Striktur nach Diphtherie *Arch klin Chir* 166 1931 pp 573-578
- Kraus (L.) Zur post diphtherischen Oesophagus Stenose *Med Klin* 1 1933 pp 351-354
- Wassermann et T. M. Turtuchino Ein Fall von ausgedehnter Diphtherie der Atmungswege und des Oesophagus *Soviet Pediatr* no 1 1935 pp 98-99
- Kiviranta (U. K.) Are any strictures of the esophagus due to diphtheria? *Ann Otol Rhinol* 58 2 juin 1949 pp 429-440

Chapter 17 Disorders Associated with Diseases of the Skin and Mucous Membranes

- Schmidt (R.) Sklerodermie mit Dysphagie *Deutsch med Wochenschr* 47 1916 p 1023
- Fessler (A.) et Pohl (R.) Sténose de l'œsophage dans la sclérodermie *Dermat Ztschr* 63 1932 pp 164-169
- Kure (K.) Yamagata (K.) Tuskada (S.) et Hiyoshi (J.) Passage Störung des Oesophagus bei Sklerodermie und Dystrophia musculorum progressiva *Klin Woch* 15 1936 p 516
- Weissenbach (H.) Sclérodermie progressive Syndrome de Thibierge Weissenbach Troubles œsophagiens *Bull de la soc franç de dermat et syphilit* 44 1937 pp 2018-2037
- Ibid* 44 1937 pp 1060-1063
- Hæsh (H.) Troubles fonctionnels et lésions de l'œsophage dans la sclérodermie *Thèse Paris* 1937
- Lindsay (J. R.) Templeton (F. E.) et Rothmanns Lesions of esophagus in generalized progressive scleroderma *JAMA* 123 1943 pp 745-750
- Richieri (A.) et d'Alotto (V.) Sclérodermie avec œsophage court et estomac thoracique *Prensa med argent* 31 15 nov 1944 pp 2331-2333
- Olsen (A. M.) O'Leary (P. A.) et Kirklin (B. R.) Esophageal lesions associated with acro sclerosis and scleroderma *Arch int med* 76 1945 pp 189-200
- Rafsky (H. A.) et Herzig (W.) Sclérodermie avec symptômes œsophagiens *Gastro Enterol* 6 1 1946 pp 35-39
- Bourne (W.) Sclerodactylie avec lésions œsophagiennes *Proc Roy Soc of Med* 40 8 juin 1947
- Nielsen (J. P.) Sklerodermie und Calcinosis cutis mit Oesophagusveränderungen und Leberschaden *Nord med* 34 1947 pp 1203-1205
- Schwarz (G. S.) et Skinsnes (O. K.) Sclérodermie progressive généralisée *The Am J Renig* 62 3 sept. 1949
- Lindsay (J. R.) Esophageal lesions in diffuse scleroderma *Laryngoscope* 59 2 83-112 1949
- Passini (C.) Aspetti endoscopici dell' esofago in un caso di acanthosis nigricans *Arch ital di otol rinol* 61 fasc 2 mars avril 1950
- Bourne (W. A.) Oesophageal lesions in sclerodactyly *Lancet* I(19) 797-798 1949
- Passini (G.) Aspects of Esophagus in a Case of Acanthosis Nigricans *Arch ital di otol & rhinol* 61(2) March April 1950
- Dornhorst (A. C.) Pierce (J. W.) and Whumster (I. W.) Oesophageal lesion in scleroderma *Lancet* I 698 1954

Chapter 18 Acute Corrosive Esophagitis (Chemical Burns)

- Belunoff La sonde à demeure dans le traitement de l'œsophagite corrosive aigue *Monats fur Ohrenh* 1940 fasc 12

- Balard Contribution à l'étude des œsophagites corrosives et de leurs séquelles Thèse Toulouse 1942
- Chalnot, Grimaud et Gosserez Lésions de l'œsophage par brûlures dues à la potasse *Ann d'oto laryngol*, no 1-2-3 janv. fév. mars 1944, p. 124
- Aubin (A) L'oblitération de l'œsophage par brûlure rétablissement du calibre après perforation guérison *Ann d'oto laryngol*, nos 1-2-3 janv. fév. mars 1944, p. 124
- Greze (A) La sonde à demeure dans les brûlures caustiques de l'œsophage *Boi de la Soc de chir de Chile*, Santiago 23, no 2, oct. 1945
- Montandon (A) Considérations sur une série de lésions de l'œsophage par des substances caustiques l'œsophage par corrosion *Practica O-R-L* 8, 5-6 II mai 1946
- Moulonguet (A) A propos des brûlures de l'œsophage *Congrès ann de la Soc belge d'Otol*, 30 juin 1946
- Uhde (G I) Brûlure chimique de l'œsophage *Ann Otol, Rhinol and Laryngol* no 4 déc 1946
- Vhoes (G I) Brûlures chimiques de l'œsophage *Ann Otol, Rhinol and Laryngol*, 55, déc 1946
- Lemarié (A) et Bilet (H) Brûlure grave œsophago-gastrique indications de la jéjunostomie *Ann d'O R-L* 13 9-10 1946, pp 462-465
- Resano (J H) Resultados alejados del tratamiento de una estrechez cicatrizal del esofago por caustico *Rev argent oto rino laryngol* 13, 3-4, 1946, pp 105-112
- Didier (G) Desorgher (G), Legrand (J) et Cadart (F) Réflexions sur un cas d'œsophagite gastrite corrosive *Soc med et anat clin de Lille* 26 fév 1947
- Montandon (A) Œsophagite par corrosion *Soc suisse O R L* 23 juin 1947
- Rebattu Rabastet et Lancon Brûlure de l'œsophage par soude caustique Hématémèse abondante au 15^e jour, anémie grave *Lyon méd* 177, 27 6 juil 1947
- Sergent (H) Brûlures des premières voies digestives par ingestion de caustiques et rétrécissement de l'œsophage *Maroc méd*, Casablanca 26 269 sept 1947, pp 303-305
- Ingmar Lagergard (Upsala) An indwelling tube of new type for the treatment of corrosive injuries in the esophagus *Acta oto laryngol*, 35 1-9, fasc 5 et 6 31 déc 1947
- Klotz (P L) Les brûlures de l'œsophage *Gaz d'hôp* Paris 120 44 1947, pp 637-641
- Didier Marroyen, Lapiere et Marsepoil (Mille) Histoire d'une œsophagite corrosive chez un enfant de trois ans *J d Sc méd de Lille*, 66 3 1 fév 1948 pp 77-79
- Fontaine et Buck (P) La gastrostomie d'urgence avec mise au repos de l'œsophage dans le traitement immédiat des brûlures de cet organe *Gaz d'hôp*, Paris 121 8-21 fév 1948 pp 101-102
- Rassudon (P M) Traitement précoce des brûlures œsophagiennes chez les enfants *Am Rev Sov Med* New York 2 avril 1948 pp 97-129
- Starck (H) Die Behandlung frischer Verätzungen der Speiseröhre durch Laugen und Säuren *Med Klin* 1948, pp 364-366
- Rodé (I) Symptômes radiologiques des lésions caustiques de l'œsophage et de l'estomac *Acta radiol* Stockholm 30 fasc 1-2, 31 août 1948
- Rebattu (J) et Martin (H) Etat actuel du traitement des brûlures et sténoses de l'œsophage *J de méd de Lyon* no 689 20 sept 1948
- Flottes Sur un cas de brûlure œsophago-gastrique par acide chlorhydrique pur *L O R I internat*, Lyon 32 9 sept 1948 pp 163-165
- Olsen (A M) Œsophagitis *Surg Gynec*, et Obst 1948 pp 372-374 In *Zentralbl für Hals Nasen und Ohren* déc 1948 p 156
- Platou (R) et Austin (G) L'œsophagite corrosive de l'enfance *New Orleans Med and Surg J* 101 6 déc 1948
- Riecker (O E) Aspects cliniques des brûlures de l'œsophage *Ar. ilische Wochenschr* Berlin 3 37-38 1948 pp 592-594
- Wiethe (C) Brûlures et lésions de l'œsophage *Monats fur Ohren* Vienne 83 4-5-6 avril juin 1949 pp 196-202
- Unterberger (S) Indications thérapeutiques nouvelles dans le traitement des brûlures récentes de l'œsophage *IV Congrès internat d'Oto-Laryngol* Londres 18-23 juil 1949
- Leary (W) Le traitement conservateur des brûlures chimiques de l'œsophage et leurs séquelles *Proc Staff Meet Mayo Clin*, 24 20 28 sept 1949 pp 506-509
- Webb (B) et Woolsey (D S) Brûlure de l'œsophage par la lessive de soude *J.A.M.A* 141 6, 8 oct. 1949 pp 384-386
- Kaiser (T) Hémorragie œsophagienne après brûlure *Wien klin Wochenschr* 61, 52 30 déc. 1949

- Cauger (G H) Associated œsophageal and gastric strictures following swallowing of caustic soda *J Lar Otol* 63 3 1949 p 154
- Fisher (G E) et Hicks (J J) La conduite à tenir dans les brûlures de l'œsophage par caustiques *Eye, Ear Nose and Throat Monthly* 28 2 1949 pp 78-83
- Shurley (W I) Les brûlures de l'œsophage par caustiques *West Virginia Med J* 45 2 1949 pp 37-41
- Surala (U) Une aide au traitement des brûlures œsophagiennes aiguës *Ann chir III gyn Fenn Helsinki* 38 fasc 3 1949
- Westerbeek (J) Brûlures de l'œsophage par la lessive de soude *Acta O R L belgica* 1 no 1, 85
- Quist Hansen Lésions de l'œsophage par agents corrosifs *Tidschr for den Norske Laeg Oslo* 69 2, 1949 pp 581-583
- Webb (B) et Woolsey (D S) Brûlures de l'œsophage par des caustiques *J A M A* no III oct. 1949
- Rosenberg (N) Kunderman (Ph J) Vroman (L) and Moolten (S E) Prevention of experimental lye strictures of the esophagus by cortisone *Arch Surg* 63(2) 147-151 1951
- Krey (H) On the treatment of corrosive lesions in the esophagus An experimental study *Acta oto-laryng* 1952 suppl 102 (49 p)
- Naegeli (Th) Zur konservativen Behandlung der Speiseröhrenverätzungen und ihrer Folgen (Conservative treatment of esophageal corrosions and their sequelae) *Helv chir Acta* 19(4-5) 324-328 1952
- Leegaard (T) Acute corrosive esophagitis *Acta chir Scand* 103(6) 481-484 1952
- Schiedt (E) Beitrag zur konservativen Behandlung der Speiseröhrenverätzungen und ihre Folgen (Conservative treatment of burns of the esophagus and their complications) *Arch klin Chir* 274(2) 113-120 1953
- Burford (T H) Webb (W R.) and Ackerman (L.) Caustic burns of the esophagus and their surgical management A clinico experimental correlation *Ann Surg* 138(3) 453-460 1953
- Leahy (L J) Caustic burns of the esophagus *Surg Gynec & Obst* 98(5) 631-633 1954
- Lawrence (G H) and Burford (T H) Caustic burns of the esophagus *Postgrad Med* 18(3) 159-164 1955
- Marchand (P) Acute caustic soda injuries of the œsophagus *S African Med J* 29(9) 195-205 1955
- Davis (M V) Early and late management of caustic burns of the esophagus *Am Surgeon* 22(3) 308-312 1956
- Carver (G M Jr) Sealy (W C) and Dillon (M L Jr) Management of alkali burns of the esophagus *J A M A* 160(17) 1447-1450 1956
- Ray (E S) and Morgan (D L) Cortisone therapy of lye burns of the esophagus *J Pediatr* 49 394-397 1956

Chapter 19 Cicatricial Stenosis

- Jung (A) et Froehlich (F) Œsophagoplastie préthoracique pour sténose cicatricielle de l'œsophage *Presse méd* fev 1935 p 307
- Soulas (A) Bougies extensibles à demeure pour le traitement de sténoses cicatricielles de l'œsophage Dilatation par le procédé du fil sans fin *Bull et mém de la Soc de chir de Marseille* séance du 25 nov 1935
- Kellemen (G) Discussion on Non malignant Obstruction of the Œsophagus *Proc Roy Soc of Med* 29 juin 1936 pp 35-38
- Bruzzi (B) De la fonction œsophagienne dans les sténoses cicatricielles *Boll mal or naso gola* no 6 1936
- Marschall Taylor (H) Aperçu préliminaire des effets que la loi sur la potasse a sur la production des strictures œsophagiennes *L O R L internat* 1936 no 2 p 123
- Kobayashi (S) Fremdkörper des Œsophagus mit narbiger Stenose *Oto Rhino Laryngol* 11 mai 1938 p 456
- Pasman Rétrécissements cicatriciels de l'œsophage *Bol y trab de la Soc de cir de Buenos Aires* no 8 8 juin 1938
- Strombeck (J P) Grave cicatricial stricture of lower esophagus treated with protheses *Steniska lak Tidning* 35 9 sept 1938 pp 1435-1438
- Hoag (L) Nouvelle méthode de dilatation graduelle des sténoses bénignes de l'œsophage *Ann d'oto laryngol* 1938 p 184
- Mounier Kuhn Contribution au traitement des sténoses cardio-œsophagiennes *Ann d'oto-laryngol* 1938 p 651

- Oliari (E) Esofagoplastica pretoracica per stenosi da ustico (nota preliminare) *Arch ed atti d Soc ital di chir* 44, 1938 pp 795-799
- Canuyt (G) et Lacroix (M) Fistule œsophago médiastinale bien tolérée chez un malade atteint de sténose cicatricielle de l'œsophage *Bronchosc, Œsophagosc et Gastros*, janv 1939, pp 19-21
- Jacques (I) L'état actuel de la question de la prévention des sténoses œsophagiennes par caustiques *Bronchosc Œsophagosc* no 1 janv 1939, pp 7-14
- Soulas (A) Documentaire clinique les maladies de l'œsophage et les sténoses cicatricielles *Soc méd chir d hop libres* séance du 3 fév 1939 in *Arch d hop*, mars 1939
- Soulas (A) Bougies extensibles et à demeure pour le traitement des sténoses cicatricielles de l'œsophage *Soc méd-chir d hop libres*, séance du 3 fév 1939, in *Arch Hospital*, no 5, mars 1939 p 295
- Schwartz (V J) Stenosis of esophagus due to lye burn intentionally induced *Lancet* 59, juin 1939, p 292
- Soulas (A) Technique du bougirage rétrograde dans les sténoses cicatricielles de l'œsophage *Bronchosc Œsophagosc et Gastros*, no 4 oct 1939, pp 199-222
- Soulas (A) Traitement des sténoses cicatricielles de l'œsophage, démonstration par film cinématographique *Ann d oto-laryngol*, 1939 p 793
- Soulas (A) Un cas de rétrécissement de l'œsophage *Ann d oto laryngol*, 1939, p 700
- Patounas (J) Un cas d'œsophagoplastie pré thoracique pour oblitération cicatricielle de l'œsophage par caustique *Revue générale du traitement chirurgical Thèse Montpellier*, 1939-1940
- Sangiovanni (V) Sulle modificazioni istologiche dell'esofago in seguito ad ingestione di caustici (ricerche sperimentali) *Arch ital di otol* 452, janv 1940, pp 1-38
- De Gouvea (Jorge) (Rio de Janeiro) L'œsophagoplastie pré thoracique dans les sténoses incurables de l'œsophage *Presse méd* no 12 fév 1940
- Belinoff (S) Un nouveau dilateur à demeure pour le traitement de l'œsophagite corrosive aigue *Bronchosc Œsophagosc et Gastros*, mars 1940
- Guisez (J) Les sténoses cicatricielles de l'œsophage peuvent-elles être guéries? *Soc méd-chir d hop libres* séance du 30 mai 1940 no 12 p 35
- Clerf (L H) Sténose par abcès tuberculeux péri œsophagien XVIII Réunion de l'Assoc amer de broncho œsophagol, New York City, 5 juin 1940
- Lot Bourniet (J A M) Sténose cicatricielle totale après brûlure Rétablissement des voies naturelles par perforation contrôlée du segment sténosé Guérison *Thèse Paris*, 1940
- Genkin (A B) Early bougirage in esophageal stenosis *Vrach Delo* 22, 1940 pp 355-358
- Izvarin (A N) Case esophageal stenosis due to ingestion of ammonia *Vrach Delo* no 9 1940 pp 617-618
- Kramann (O) Formation d'un nœud sur une bougie dilatatrice dans l'œsophage *Rev de laryngol* 1940 p 354
- Popian (St) Cicatrices vicieuses et les sténoses hypo pharyngo œsophagiennes à la suite d'ingestion de liquide caustique *Rev de laryngol*, 1940, p 356
- Etienne Lapevrie Passebois et Patounas Rétrécissement (oblitération totale) à la suite de brûlure par caustique reconstruction d'un œsophage anté thoracique Présentation d'un malade *Soc des Sc méd de Montpellier* séance du 24 janr 1941, in *Montpellier méd*, no 90-92 mars avril 1941
- Brémond A propos des rétrécissements cicatriciels de l'œsophage *Soc de méd de Marseille*, 4 mars 1941 in *Presse méd* no 24 20 avril 1942 p 313
- Belinoff (S) Peut-on prévenir la stricture par corrosion de l'œsophage? *Wien med Wochenschr* 91 14 15 avril 1941, p 275
- Modet (L) Rétrécissement cicatriciel de l'œsophage par déchirure de sa tunique musculaire au cours d'une attaque de hoquet, auto-observation. *Revue française de médecine et de chirurgie* 4 35 juin 1941
- Dick La runnellisation des strictures infranchissables de l'œsophage et de ses oblitérations œsophagoplastie anté thoracique *Zentralbl für Chir* 68, 45 8 nov 1941
- Rozzi (E) Atresia cicatriziale de l'esofago da infezione tífica Di un accorgimento tecnico per la delimitazione della porzione interessata *L O R I ita'* 1941
- Rocher (H L) et Despons A propos d'un rétrécissement cicatriciel de l'œsophage compliqué d'abcès du poulmon Fistule œsophago bronchique deux gastrostomies Dilatation par oliviers métalliques sur fil sans fin (méthode du P^r Portmann) Guérison *Soc de méd infant de Bordeaux et du Sud Ouest* séance du 14 janv 1942 in *Bull et mém de la Soc de méd infant de Bordeaux et du Sud Ouest* no 2, 1942 pp 23-24
- Soulas (A) Traitement des rétrécissements cicatriciels de l'œsophage Danger de certains

- procédés *Bull et mém de la Soc de méd de Paris* no 2 séance du 24 janv 1942 pp 53-58
- Aubriot (P) et Coulet (G) Sténose cicatricielle de l'œsophage par absorption d'alcalis caustiques *Soc de méd de Nancy* séance du 28 janv 1942 in *Rev méd de Nancy* 15 fév 1942 pp 102-105
- Drash (P) et Woodward (I) Le traitement des rétrécissements infranchissables de l'œsophage par une voie d'approche combinée et extra-œsophagienne *Surgery* 7 6 juin 1942
- Struben Les strictures de l'œsophage par caustiques alcalins *Nederl tijdschr voor geneesk* 86 49 5 déc 1942
- Bouderbala (A) Du cathétérisme à l'endoscopie dans les sténoses de l'œsophage essai de traitement des sténoses par brûlures de l'œsophage par l'étincelage et la dilatation *Thèse Paris* 1942
- Le Mée et Soulas (A) Communication œsophago trachéale et médiastinale consecutive à une sténose cicatricielle de l'œsophage *Soc de laryngol d'hop de Paris* séance du 21 juin 1943 p 114
- Franzas (F) (Helsingfors) Rétrécissement du tube digestif après absorption de liquides caustiques en Finlande *Acta chir Scand* 89 suppl 81 1943
- Lindsay Jr Templeton (F E) et Rothmann (S) Lesions of esophagus in generalized progressive scleroderma *J A M A* 123 1943 pp 745-756
- Lemaître et Rancoule Sténose par bride fibreuse simulant un cancer de la bouche œsophagienne *Ann d'oto laryngol* Paris nos 1-2-3 janv fév mars 1943
- Guisez (J) Des sténoses graves non cancéreuses du tiers inférieur de l'œsophage *Paris méd* no 7 10 avril 1944 p 128
- Gueullette (R.) Œsophagoplastie pré thoracique pour rétrécissement cicatriciel incurable de l'œsophage *Mém de l'Acad de chir* 70 33 bis séance du 20 déc 1944 p 448
- Yudin (S S) Surgical construction of 80 cases of artificial esophagus *Surg Gynec & Obst* 78 1944 p 561
- Gueullette (R) Étude fonctionnelle d'une œsophagoplastie pré thoracique *L O R L internat* avril 1945
- Cueullette (R) Traitement d'une sténose totale de l'œsophage par création d'un œsophage pré thoracique *Soc de laryngol d'hop de Paris* séance du 18 juin 1945
- Johow (A) Transplantation pré thoracique de l'estomac dans le tissu cellulaire sous cutané pré thoracique pour sténose œsophagienne opération de Kirschner *Bol Soc de chir de Chile* Santiago 23 1 juin 1945
- Mayoux (M) et Dumarest (J) Sténose œsophagienne d'aspect cicatriciel de nature cancéreuse métastatique *Ann d'oto laryngol* 1944-1945 no 1 juil sept 1945
- Clark (D E) et Adams (W E) Œsophago gastrostomie trans thoracique pour sténose bénigne de l'œsophage inférieur *Ann Surg* 122 6 déc 1945 pp 942-953
- Machado Ortega (R) Sténose œsophagienne de l'enfance *Arch de méd inf* 14 4 1945 pp 279-284
- Cojazzi (Luigi) Il trattamento dietroscopico in 50 casi di stenosi esofagea da caustici *Atti d Soc med chir di Padova e di Fac di med e chir d Univers di Padova* 24 1945
- Olsen (A M) O Leary (P A) et Kirklin (B R) Esophageal lesions associated with acrosclerosis and scleroderma *Arch Int Med* 76 1945 pp 189-200
- Mallet Guy (P) et Marion (P) Œsophagoplastie pré thoracique pour oblitération cicatricielle *Soc de chir de Lyon* séance du 25 janv 1946 in *Lyon méd* no 15 14 avril 1946 p 235
- Longmire Jr (W) et Ravitch (M) Une nouvelle méthode pour construire un œsophage artificiel *Ann Surg* Philadelphie 123 1 janv 1946
- Schuermans (J) et Titeca (J) Fausse anorexie mentale par rétrécissement cicatriciel méconnu de l'œsophage *J belge de neurol et de psych* 44-45-46 1 janv 1946 pp 61-66
- Flynn (R H) Rétrécissement de l'œsophage *Med J Austral* 28 sept 1946
- Despons (J) Une calamité du moment les rétrécissements cicatriciels de l'œsophage chez l'enfant *J de méd de Bordeaux et du Sud Ouest* 123 17-18 sept 1946
- Sweet (R H) Œsophagectomie subtotal avec anastomose œsophago gastrique intra thoracique très haute pour le traitement d'une sténose cicatricielle très étendue *Surg Gynec & Obst* 83 4 oct 1946 pp 417-427
- Soulas (A) Dilatation des sténoses des bronches et de l'œsophage sous courant galvanique Présentation de l'appareil *Soc de laryngol d'hop de Paris* séance du 16 nov 1946
- Sudaka (P) Les rétrécissements cicatriciels de l'œsophage *Cahiers méd de l'Union franç* no 4 nov 1946 pp 251-258
- Gallart Mones (F) et Romero (A) Sténose de l'œsophage par ulcère probable *Med Espan* Valence 15 93 nov-déc 1946

- Guisez (Jean) Formes peu connues de sténoses graves de l'œsophage *Soc de méd de l'ars* séance du 27 déc 1946
- Latraverse (V) Les sténoses de l'œsophage *L'Union méd du Canada*, Montréal 69 2 sept In *J de chir* 62, 7-8 1946
- Ilroyles (E N) Nouveau dilateur œsophagien *Ann des mal de l'or* 1946 p 467
- Davis (J S) et Stafford (I) Construction réussie d'un œsophage extra thoracique *Bull Johns Hopkins Hosp* Baltimore, 67, 4 oct In *J de chir* 62 7-8, 1946
- Rafsky (H A) et Herzog (W) Sclérodémie avec symptômes œsophagiens *Gastro-Enterol*, 6, 1, 1946 pp 35-39
- Aubin (A) et Clerc (P) Quelques cas difficiles de rétrécissement de l'œsophage *Ann Oto-Laryngol*, 13 9-10 1946 pp 448-452
- Soulas Aboulker et Autier (Ch) Fistule œsophago pulmonaire dans une sténose cicatricielle de l'œsophage *Ann d O R-L* 13 5-6 1946
- Soulas (A) Sténoses cicatricielles de l'œsophage qu'on pourrait croire totales Discussion thérapeutique *Ann d O R-L*, 13 5 6 1946
- Autier Traitement des rétrécissements cicatriciels de l'œsophage par fil sans fin *Thèse l'ars* 1946
- Arrighi Sur un cas de rétrécissement cicatriciel de l'œsophage *Algérie méd*, Alger 51, 6 juin juil 1947
- Jacob (Al) Sténose œsophagienne consécutive à des brûlures par ingestion d'acide azouque *Lyon méd*, 177 27, 6 juil 1947
- Jeker (L) (Bale) Contribution à la plastie anté thoracique de l'œsophage *Schir méd Wo fens* 77 27-28 12 juil 1947 pp 742-745
- Longmire (W P) Une modification de la technique de Roux pour la reconstruction anté thoracique de l'œsophage *Surgerg* 22 1, juil 1947 pp 94-100
- Torre (P) Les rétrécissements de l'œsophage par caustique *L'Afrique franç chir* Alger 5, 4 juil aout 1947
- Prohaska (J V) et Sloan (J) L'emploi du jéjunum pour créer un œsophage artificiel anté thoracique *Surgery* 22 2 aout 1947 pp 191-197
- Cojazzi (Luigi) et Gurian (Gaetano) La sindrome periesofagea nelle stenosi da caustici *Estrat d riv margin otolaryng* 5, fasc 3, sept 1947
- Sergent (H) Brûlures des premières voies digestives par ingestion de caustiques et rétrécissements de l'œsophage Le traitement endoscopique et ses résultats *Maroc méd* no 269 sept 1947 pp 303-305
- Paris (J) A propos d'un cas de rétrécissement cicatriciel de l'œsophage d nature syphilitique très probable *Soc de méd du Nord* 31 oct 1947
- Autier (Charles) 68 cas de rétrécissements de l'œsophage *Ann d oto laryngol*, 64 10 oct 1947
- Ferrand (J) et Gardel (J) Œsophago gastrostomie transpleurale droite pour rétrécissement cicatriciel de l'œsophage thoracique *Soc de chir d Alger* 28 nov 1947
- Nadal et Piéquet Sténoses œsophagiennes post radiothérapiques *Soc d électro-radiol méd de France* 9 déc 1947
- Jones (H) Traitement d'une sténose de l'œsophage chez un enfant ayant avalé de la soude *O R L internat* déc 1947 p 191
- Pape (R) Sur les complications typiques de la sténose de l'œsophage *Wien Zeitschr für inn Mediz* 27 fasc 6 juin In *J de chir* 63 9 1947
- Filipo (D) Stenosi esofagea de caustici *Atti clinica O R L di Roma* 1947
- Dussillol Sur un cas de sténose cicatricielle de l'œsophage *Rev d O R L* 66 1947
- Lippa (B) Contribution au traitement des sténoses cicatricielles de l'œsophage *Ann d oto-laryngol* 1947 p 228
- Stevenson (Th) La reconstruction de l'œsophage par un tube à fare interne cutanée *Surg Gynec & Obst* 64 2 fev 1947
- Hunermann Médiastrinite après traitement des sténoses cicatricielles de l'œsophage et autres interventions de l'œsophage *Woch Festsch Bad Worishof* 1, 6-7 pp 138-141 1947
- Sydney (G S) et Holt (L L) Prophylaxie des sténoses patassiques de l'œsophage *Ann d oto-laryngol* 64 7, 1947 p 381
- Soulas (A) Les rétrécissements cicatriciels de l'œsophage et le bougierage rétrograde *Sem d hôp de Paris* no 11 21 mars 1947 pp 677-682
- Charterjee (S C) Rétrécissement de l'œsophage après une fièvre typhoïde *Indian J Med* Calcutta 14 53 1947 pp 13-15
- Gueullette La question de l'œsophagoplastie (A propos du film de Judine) *Acad de chir* 21 janv 1948

- Pueston (C B) et Stephen (C J) Résection de l'œsophage pour sténose cicatricielle *Arch Surg* 36 1 janv 1948, pp 34-47
- Rebattu et Gaillard Deux cas de rétrécissements cicatriciels de l'œsophage avec accidents graves au cours de la dilatation Guérison par la pénicilline *Soc de laryngol de Paris* 15 mars 1948 in *Ann d oto laryngol* 65 10 pp 622-624
- Szénès (H) Sténose de l'œsophage à étiologie spéciale *Soc autrich d O R L* séance du 2 déc 1947 in *Monats fur Ohrenh und L R H* 3 mars 1948
- Baumann (J) Rétrécissement de l'œsophage traité par résection *Lekarske Listy* Brno 3 9 1 mai 1948
- Kucera Le rétrécissement de l'œsophage après corrosion *Lekarske Listy* Brno 3 9 1 mai 1948
- Creyx (M) Laumonier (P) Lévy (J) Texier (L) et David Chaussé (J) Diagnostic étiologique liguéux d'une sténose œsophagienne (syphilis probable) *Soc de méd et de chir de Bordeaux* 7 mai 1948
- Gignoux (M) Rétrécissement cicatriciel de l'œsophage et du pylore par caustique Gastro entéro anastomose Guérison *Soc d O R L de Lyon et de la Région* séance du 23 mai 1948
- Majoux Neuf cas de sténose cicatricielle de l'œsophage *Soc d O R L de Lyon et de la Région* 23 mai 1948
- Aloumier Kuhn Rétrécissement grave cicatriciel de l'œsophage avec double fistule œsophago aénienne *Soc d O R L de Lyon et de la Région* 23 mai 1948
- Bradbeer (W H) Sténose non maligne de l'œsophage *Soc de laryngol d hop de Paris* séance du 24 mai 1948
- Vaspetoff Orsoni Hacker et Gachiniard Rétrécissement de l'œsophage récidivant puis inserré Dilatation sur fil sans fin *Soc de laryngol d hop de Paris* séance du 24 mai 1948
- Gross (R F) Traitement d'un rétrécissement serré de l'œsophage par œsophagectomie partielle et reconstruction de l'œsophage par anastomose terminale *Surgery* 23 5 mai 1948 pp 715-744
- Mei (G M) Nouvelle méthode d'œsophagoplastie *J Internat Coll Surgeons* 1 3 mai juin 1948 pp 210-277
- Schmidt (H W) Le rétrécissement cicatriciel de l'œsophage *Proce Staff Meet Mayo Clin Rochester* 23 12 9 juin 1948 pp 265-267
- Martinez Gomez (S) Sténose œsophagienne consécutive à une médiastinite plastique *Med espan* Valence 11 112 juil 1948
- Karatay (S) Sténose totale de l'œsophage due à une brûlure par ingestion de lessive *Med Oto Laryngol* Chicago 48 1 juil 1948 pp 83-85
- Gottsmann (R) Sur 10 cas d'œsophagoplastie pré thoracique *Ret de chir de Sao Paulo* 14 1 juil août 1948
- Gignoux (M) et Colson (P) Rétrécissement cicatriciel de l'œsophage et du pylore par caustique Dilatation Gastro entéro anastomose Guérison *J de méd de Lyon* no 689 20 sept 1948
- Rebattu (J) et Martin (H) Etat actuel du traitement des brûlures et sténoses de l'œsophage Résultats thérapeutiques *J méd Lyon* 29 689 20 sept 1948 pp 643-647
- Keeley (C) Sténose bénigne de l'extrémité inférieure de l'œsophage traitée par résection et anastomose œsophago gastrique *Canad Med Assoc J* Montréal 59 4 oct 1948
- De Bekey et Oschner Chirurgie des sténoses bénignes de l'œsophage *Int Surg Dig* 46 oct 1948 p 195
- Lambert Autier et Robichon Lésions bucco dentaires observées au cours des rétrécissements cicatriciels de l'œsophage *Rev d stomatol* 49 10 oct 1948 pp 633-639
- Benedict (E B) et Sweet (R H) Rétrécissement bémun de l'œsophage *Gastro-Entérol* 11 5 nov 1948 pp 618-629
- Giangrasso (A) Sur un cas de sténose extrinsèque de l'œsophage juxta cardiaque post inflammatoire *Ann ital di chir* Bologne 25 fasc 12 déc 1948
- Bolstad (D S) Pyloric obstruction and stricture of the œsophagus following ingestion of lye (Sodium hydroxide mixed with sodium carbonate) *Arch Oto Laryngol* 47 1948 p 180
- Cody (C C) Management of œsophageal strictures *Arch Oto Laryngol* 47 1948 p 133
- Mayer (J) Sekundärer Kardiospasmus diagnostiziert durch retrograde Gastro œsophagoskopie *Monats Ohrenh* 87 1948 p 137
- Claggett (O T) et Schmidt (H W) Le traitement chirurgical de la sténose acquise de l'œsophage avec fistule œsophago-bronchique et bronchiectasie du poulmon droit dans son entier *Surgery* 1948 13 2 1948 pp 221-226
- Messerklinger (W) Meningitis und Hirnabszess als Komplikation bei Bougerung wegen Laugenstenose des Oesophagus *Woch Ohrenh Wien* 87 11 1948 p 473

- Autier (Charles) Sténoses cicatricielles de l'œsophage, passage du fil par voie rétrograde
Bull Soc de broncho-œsophagol et de gastroc de langue franç, 1948
- Hartenau (W.) Aussergewöhnlicher Verlauf einer Laugenstenose Mischr Ohren heilk 87
1948, pp 464-466
- Nagel (O.) Ösophagusstenosen nach primären Paraffinoplonben Tuberkulose-arch 1948
pp 242-250
- Ungerecht (K.) Die Sondenbehandlung der Ösophagusnarbenstenosen mittels Faden und deren Schwierigkeiten *H N O, Beih z Z Hals usw Heilk* 1, 1948 p 190
- Szenes (H.) Sténose œsophagienne d'origine embryonnaire *Monats fur Ohrenh und Laryng Rhinol Vienne* 82, 4, 1948 pp 166-171
- Ludin (V. M.) Sténose de la partie inférieure de l'œsophage par modification relevant d'une œsophagite aigue *Acta radiol Stockholm*, 30, 176-177 1948, pp 362-364
- Möbling Tube à air comprimé nouvelle méthode de traitement des sténoses œsophagiennes
H N O, Berlin 1/3 1948 p 135
- Wirth (J.) Dilatation des sténoses œsophagiennes et nouvelle méthode d'introduction du fil sans fin *Pediatrica Danubiana, Budapest* 3 4 1948 pp 181-183
- Sudaka (P.) et Gardel (J.) A propos du traitement des sténoses cicatricielles serrées ou infranchissables de l'œsophage *Cal ers néd de l'Union fran*, 4 26 janv 1949 pp 31-34
- Monod (R.) et Germain (A.) Rétrécissement traumatique de l'œsophage cervical datant de 15 ans traité avec succès par œsophagoplastie pré-thoracique avec anastomose entre une anse jejunaie et un diverticule pharyngo œsophagien sus-structural *Acad de chir* 9 fév 1949
- Chalnot et Lochard Anastomose intra thoracique œsophago gastrique pour sténose cicatricielle de l'œsophage au niveau de la crosse de l'aorte *Soc de Méd de Nancy*, 9 fév 1949
- Jeziuro (Z.) Trois cas de plastie anté thoracique *Polski Przegląd Chir*, Varsovie 21 4 janv fév 1949
- Denecke (H. J.) L'image œsophagoscopique en cas de sténose œsophagienne après médisinuite couenneuse par huile de paraffine *Arch fur Ohren Nasen und Kehlkopf Heilk Berlin* 135 4 26 mars 1949 pp 503-504
- Brain (R.) Structures simultanees de l'œsophage et de l'estomac *Brit J Surg* 36 144 avril 1949
- Lortat Jacob (J. L.) et Duperrat Sténose inflammatoire de l'œsophage traitée par la résection
Soc anat de Paris, 5 mai 1940
- De Vernejoul Œsophagoplastie par le procédé de Lever *Soc de chir de Marseille* séance du 12 mai 1949
- Ruedi (R.) Traitement chirurgical de la sténose cicatricielle de l'œsophage *Bull d'r Scher 1 er Akad d'r Medizin Wissensch Bale* 5 2-3, mai 1949 pp 114-131
- Paulino (F.) (Rio de Janeiro) Sténose cicatricielle de l'œsophage Œsophagectomie *Rev med do Parana* 18, 3, mai juin 1949 pp 131-135
- Harrison (A. W.) Œsophagoplastie intrathoracique d'intestin grêle dans les sténoses hautes de l'œsophage *J Surg* 18 3 juin 1949 pp 316-326
- Taylor (H.) Etude d'une opération d'œsophagostomie avec œsophago-gastrostomie pré sternale *Brit J Surg* 36, 144 1949 pp 419-420
- Manuelyan (B.) Dilatation rétrograde d'une sténose totale de l'œsophage *Türk Tip Cemittii Mecmuası Istamboul* 5 6 juin 1949
- Dumont (P. U.) (Vendôme) Trois cas de guérison de sténose œsophagienne par dilatation rétrograde *Soc de br œsoph et de gastr de Langue franç* 10 juil 1949
- Bloch (André) Sur le traitement des rétrécissements cicatriciels de l'œsophage par caustique chez l'enfant *IV Congrès internat d'Oto Laryngol Londres* 18-23 juil 1949
- Ferreri (G.) Sur la dilatation progressive rétrograde de l'œsophage *II Congrès internat d'Oto Laryngol Londres* 18-23 juil 1949
- Danielewicz (J.) Traitement chirurgical du rétrécissement cicatriciel d'œsophage œsophagotomie interne avec séparation III laide la de cellophane *II Congrès internat d'Oto-Laryngol Londres*, 18-23 juil 1949
- Lewentisz Wojnarowski (Lodz) Diathermo-coagulation avec une électrode en spirale comme traitement de l'étranglement de l'œsophage *II Congrès internat d'Oto-Laryngol Londres* 18-23 juil 1949
- Rubin (H. J.) Nouvelle méthode de dilatation sur fil dans les rétrécissements de l'œsophage *The Laryngoscope* 1949 59 12 1361-1364
- Wells (C. A.) Howell Edwards et Marcus Perforation consécutive à la dilatation des sténoses de l'œsophage *Thorax* juin 1949
- Kalocsay (C.) (Budapest) Quelques cas intéressants de sténose œsophagienne *II Congrès internat d'Oto-Laryngol Londres* 18-23 juil 1949

- Koleszar (G) (Budapest) Les rétrécissements de l'œsophage causés par les caustiques et leur traitement *II Congrès internat d'Oto Laryngol* Londres 18-23 juil 1949
- Soulas (A) Statistique de 20 ans d'expérience des rétrécissements de l'œsophage thoracique d'origine non cancéreuse *IV Congrès internat d'Oto Laryngol* Londres 18-23 juil 1949
- Da Costa Quinta (A) Sonde dilatatrice pneumatique pour le traitement des sténoses bénignes de l'œsophage *II Congrès internat d'Oto Laryngol* Londres 18-23 juil 1949
- Tucker (G) Terracol (J) et Gerlings (P. G.) Les rétrécissements non néoplasiques de l'œsophage et leur traitement *IV Congrès internat d'Oto Laryngol* Londres 18-23 juil 1949
- Martinho da Rocha (R.) Sur un cas de sténose de l'œsophage par ingestion de soude caustique *Brasil Medico* Rio de Janeiro 63 34/35 20 et 27 août 1949 pp 220-223
- Taylor (E. R.) et Clagett (O. T.) (Rochester) Un cas de sténose œsophagienne par caustique résection de l'œsophage avec œso-gastrostomie cervicale *Proc Staff Meet Mayo Clin* 24 18 31 août 1949 pp 473-476
- Feldman (M.) Leukoplakiaktratos of the esophageal stricture *Gastro-enterol* 13 2 août 1949, pp 175-78
- Schwartz (G. S.) et Skinsnes (O. K.) Sclérodémie progressive généralisée *Am J Rontg* 63 3 sept 1949
- Ragnell (A.) La reconstruction de l'œsophage *Acta chir Scand* Stockholm 18 3 à 6 3 sept 1949
- Olivier (Cl) et Faurel (J.) Œsophagoplastie préthoracique par le procédé de Judine *Acad de chir* séance du 12 oct 1949
- Olivier (M.) Œsophagoplastie pré thoracique au moyen d'une anse grele pour sténose cicatricielle de l'œsophage *Acad de chir* 12 oct 1949
- Autier (M.) Un cas compliqué de sténose cicatricielle de l'œsophage *VI II Congrès franç d'oto laryngol* 20 oct 1949
- Fritz (K.) Œsophagustenosen seltener Genese *Mtschr Ohrenh* 83 10-12 oct déc 1949
- Fracasso (E.) et Gurian (G.) Les aspects radiologiques des altérations perœsophagiennes dans la sténose par caustiques *Radiol med* Turin 35 11 nov 1949
- Barraya (M.) Rétrécissement cicatriciel de l'œsophage *Acad de chir* 14 dec 1949
- Lortat Jacob (J. L.) Un procédé d'œsophagoplastie utilisant l'estomac *Presse méd* 31 dec 1949
- Santy et Aubin La suppression par plastie de la poche sus structurale d'un rétrécissement thoracique de l'œsophage *Ann oto-laryngol* Paris 66 6 1949 p 305
- Lindsay (J. R.) Esophageal lesions in diffuse scleroderma *Laryngoscope* 59 2 1949 p 83
- Boshnack (M.) et Jones (D. H.) Rétrécissement de l'œsophage rapport d'un cas *Eye Ear Nose and Throat Monthly* 78 6 1949 pp 269-272
- Kiviranta (U. K.) Les rétrécissements de l'œsophage sont ils dus en quoi que ce soit à la diphtérie? *Duodecim* Helsinki 65 1 1949 pp 67-77
- Niero (C.) et Zaffagnini (V.) La sténose de l'œsophage par brûlures caustiques *Arch ital di chir* Bologne 72 1 1949
- Niero (G.) La sténose totale de l'œsophage par ingestion de caustiques *Arch ital di chirurgia* 72 2 1949 pp 69-90
- Hanson (H. E.) Ein Fall von Œsophagusplastik nach Judine *Nord med* 1949 p 552
- Gaiger (G. H.) Clinical record Associated œsophageal and gastric strictures following swallowing of caustic soda *J Laryng. & Otol* 63 1949 pp 151-158
- Niero (G.) La sténose totale de l'œsophage par ingestion de caustiques *Arch ital di chir* Bologne 72 2 1949
- Leroux (Michel) Contribution à l'étude du traitement des sténoses cicatricielles et complètes de l'œsophage par voie trans thoracique *Thèse Nancy* 1949-1950
- Santy (P.) et Maillet (P.) Quatre cas d'anastomose œso-gastrique sus aortique pour sténose cicatricielle haute de l'œsophage *Lyon chir* 45 2 fev mars 1950
- d'Allaines (F.) Sur le traitement de la sténose cicatricielle de l'œsophage *Acad de chir* 18 fev 1950
- Soulas (A.) et Autier (Ch.) La gastrostomie en vue de l'œsophagoscopie par voie basse et du traitement des rétrécissements cicatriciels de l'œsophage *Soc de broncho-œsophagol et de gastrosc de langue franç* 14 mai 1950
- Hepp (J.) Les rétrécissements cicatriciels de l'œsophage *Acad de chir* 18 janv 1950
- Strauss (G. D.) Stricture de l'œsophage associée à une opération sur le duodénum *Arch Oto Laryngol* 51 11 fév 1950
- Bugden (W. F.) Résection de l'œsophage pour sténose par caustique *New York State J Med* 100 15 mars 1950

- Lortat Jacob (L.) et Duperrat Sténose inflammatoire de l'œsophage traitée par la résection *Pathologie Paris* no 12 janv 1950, pp 13-15
- Ballivet L œsophagoplastie pré thoracique avec anastomose directe œsophago-jéjunogastrique quatre cas chez l'enfant *Acad de chir* 10 janv 1950
- Puyo (R.) et Babakr (F.) Sténose spasmodique de l'œsophage *Rev de laryngol* : Bordeaux 71 1-2 janv fév 1950
- Hubbard (T B.) et Leven (N L.) Dilatation de l'œsophage Une contre indication à l'utilisation du fil dégluti Nouveau procédé *Surgery*, 77 1 janv 1950
- Mattos Barretto Traitement de l'œsophagite corrosive, des sténoses et atrophies cicatricielles de l'œsophage *Ann d oto laryngol*, 67, mai juin 1950
- Teenders (H.) Behandlung und Behandlungserfolge der Speiseröhrenverätzungen (Treatment and results in caustic lesions of the esophagus) *Beitr klin Chir* 179(3) 385-394 1950
- Hepp (J.) A propos des rétrécissements cicatriciels de l'œsophage (Treatment of cicatricial stenosis of the esophagus) *Mém Acad chir Paris*, 76(1-2-3) 84-86 1950
- Rapant (V.) et Hromada (J.) Surgical treatment of corrosive stenoses of the thoracic part of the esophagus by a singlestage palliative anastomosis Intracervical retrovascular anastomosis without resection of the esophagus *J Thoracic Surg* 20(3) 454-473 1950
- Valdoni (P.) Traitement radical des sténoses œsophagiennes La prévention de l'œsophagite peptique post-opératoire (Radical treatment of stenosis of the esophagus Prevention of postoperative peptic esophagitis) *Presse méd*, 59(49) 1216-1218, 1951
- Rock (T.) Sarnelli (V.), Ventrella (A.) and Negro (L.) Il trattamento delle stenosi esofagee cicatriziali da caustici (The treatment of cicatricial esophageal strictures due to caustics) *G ital chir* 8(3) 173-187 1952
- Aves (F H.) Desforger (G.) and Strieder (J W.) Mercury bag bolus to expedite passage of esophageal dilator guide string *J Thoracic Surg* 24(1) 34-37 1952
- Kumantova (N N.) Lecenje posledstvi ozogov i profilaktika suzenij pisevodov (The treatment of corrosive injuries and the prophylaxis of stenosis of the esophagus) *klin Med (Mosk)* 31(2) 60-64 1953
- Rosenberg (N.) Kunderman (P J.) Vroman (L.) and Moolten (S F.) Prevention of experimental esophageal stricture by cortisone II Control of suppurative complications by penicillin *Arch Surg* 66(5) 593-598 1953
- Holinger (P H.) Johnston (K C.) Potts (W J.) and DaCunha (I.) The conservative and surgical management of benign strictures of the esophagus *J Thoracic Surg* 28(4) 345-366 1954
- Emerson (E B Jr.) Intraluminal resection of esophageal strictures *Arch Surg* 71(6) 863-866 1955
- MacLean (L. D.) and Wangenstein (O H.) The surgical treatment of esophageal stricture *Surg Gynec & Obst* 103(1) 5-14, 1956
- Douglas (W K.) Esophageal strictures associated with gastroduodenal intubation *Brit J Surg* 43(180) 404-409 1956
- Wyndham (N S.) The significance of gastric mucosa in the esophagus *Brit J Surg* 43(180) 409-412 1956
- Perrotti (G.) Trattamento delle stenosi esofagee alte ed estese da caustici con anastomosi esofago gastrica cervicale transtoracica sinistra semplificata *Riforma med* 1 1956
- Nardi (G L.) Surgical treatment of lye strictures of the esophagus by mediastinal colon transplant without resection *New England J Med* 256(17) 777-780 1957

Chapter 20 . Esophageal Substitution

- Hantahan (E M.) The use of a migrated skin flap for both lining and covering of an artificial antethoracic esophagus *Plastic and Reconstructive Surg* 3(5) 474-480 1947
- Van Prohaska (J.) and Sloan (J.) The use of the jejunum in the construction of an antethoracic esophagus *Surgery* 22(2) 191-197 1947
- Ragnell (A.) Reconstruction of the esophagus *Acta chir Scand* Stockholm 98(3-6) 369-384 1949
- Lorenz (O.) Die Wiederherstellung der Speiseröhre unter Brucknechtigung eines einfachen Verfahrens der Hautschlauchbildung (The reconstruction of the esophagus with consideration of a simple procedure of skin tube construction) *Med Monatsschr Stuttgart* 3(5) 375-380 1949
- Bricker (E M.) Burford (T H.) and Euseman (B.) The use of tubed pedicle grafts in carcinoma of the upper esophagus *J of Thoracic Surg* 18(3) 304-315 1949
- Chalnot (P.) and Stehlin (H F.) Au sujet du fonctionnement des bouches d'œsophago-

- gastrostomie après résection œsophagienne (The functioning of esophago gastric anastomoses after esophagectomy) *J de radiol et d'electrol et Arch d'electricité méd* Paris 30(11-12) 647-650 1949
- Petrov (B. A.) and Khundadze (G. R.) A new aid to additional mobilization of the small intestine for formation of an artificial esophagus) *Khirurgiya* Moscow 1 57-66 1950
- Monod and Broca (Ph.) Œsophagoplastie préthoracique palliative avec le colon transverse pour cancer inopérable de l'œsophage thoracique (Palliative prethoracic esophagoplasty using the transverse colon for inoperable carcinoma of the thoracic esophagus) *Mém Acad chir* Paris 76(28-29) 824-828 1950
- Dubost (Ch.) and Bernier (L.) Un procédé d'allongement de l'anse intestinale au cours de l'œsophagoplastie préthoracique (A method for lengthening the intestinal loop during prethoracic esophagoplasty) *Rev chir* Paris 69(7-8) 193-197 1950
- Ballivet L. œsophagoplastie préthoracique avec anastomo = directe œsophago jéjuno gastrique (4 cas chez l'enfant) (Prethoracic esophagoplasty with direct esophago jejuno gastrostomy [4 cases in children]) *Mém Acad chir* Paris 76(1-2-3) 73-84 1950
- Shaw (M. H.) Reconstruction of the esophagus *Brit J Plast Surg* 3(2) 96-107 1950
- Orsoni (P.) and Toupet (A.) Utilisation du colon descendant et de la partie gauche du colon transverse pour l'œsophagoplastie pré thoracique (Use of the descending colon and the left portion of the transverse colon for the performance of a prethoracic esophagoplasty) *Presse méd* 58(44) 804 1950
- Picot Roux (L.) Epicrise de la première opération de l'œsophagoplastie préthoracique Faite par le Dr César Roux en 1906 Partie Chirurgicale Œsophago jéjuno gastrostomose (Follow up of the first endothoracic esophagoplasty performed by César Roux in 1906 Surgical part Esophago jejuno gastrostomy) *Rev. méd de la suisse romande* Lausanne 70(1) 19-28 1950
- Rudler (J. C.) et al Enquête chirurgicale sur 28 œsophagoplasties préthoraciques avec le colon pour cancer de l'œsophage (Twenty eight presternal esophagoplasties with colon for cancer of esophagus) *Rev. chir* Paris 70(4-8) 193-205 1951
- Orsoni (P.) and Lemaire (M.) Technique des œsophagoplasties par le colon transverse et descendant (Technique of esophagoplasty using transverse or descending colon) *J chir* Paris 67(6-7) 491-505 1951
- Lazar (D.) Formation of a pre thoracic œsophagus A recent modification *J int chir* 11(2) 147-151 1951
- Lortat Jacob (J. L.) Œsophagoplastie isopéristaltique transthoraco médiastinale avec le colon transverse (Transthoracomediastinal isoperistaltic esophagoplasty with the transverse colon) *Mém Acad chir* Paris 77(18-19) 586 1951
- Villemain Dufour Rigaud and Cabanué (H.) Les facteurs anatomiques conditionnant l'ileo colo œsophagoplastie pré thoracique (Anatomical factors governing prethoracic ileo colo-esophagoplasty) *Bordeaux chir* 2 49-55 1951
- Goumain and Cabanué (H.) Les techniques d'œsophagoplastie préthoracique (Techniques for prethoracic esophagoplasty) *Gaz hop* Paris 124(49) 581-583 1951
- Lafargue (P.) Dufour (R.) Cabanué (H.) and Chavannaz (J.) L'ileo colo œsophago plastie pré thoracique (Technique et application au traitement du cancer œsophagien) Œsophagoplastie préthoracique à l'aide du colon droit et de l'iléo terminal Iléo colo-œsophagoplastie (Prethoracic esophagoplasty [technique and application in the treatment of esophageal cancer] Prethoracic esophagoplasty using the right colon and the terminal ileum) *Bordeaux chir* 1 5-15 1951 *Mém Acad chir* Paris 77(12-13) 362-372 and 77(14-15) 420-422 1951
- Rudler and Monod Broca (Ph.) Un cas d'œsophagoplastie palliative retro sternale avec l'iléo colon droit (A case of palliative retro sternal esophagoplasty using the terminal ileum and right colon) *Mém Acad chir* Paris 77(22-23-24) 747-749 1951
- Orsoni (P.) Contribution à l'étude de l'œsophagoplastie dans le traitement palliatif du cancer de l'œsophage Réflexions sur 7 cas d'œsophagoplastie pré thoracique par anse grêle sans raccord cutané (A study on the palliative treatment of carcinoma of the esophagus by esophagoplasty) *Mém Acad chir* Paris 77(27-28) 876-885 1951
- Orsoni (P.) Possibilités nouvelles dans la chirurgie de l'œsophage grâce à l'utilisation du colon transverse pour l'œsophagoplastie (New possibilities in esophageal surgery from utilization of the transverse colon for esophagoplasty) *Presse méd* 59(14) 272 1951
- Berman (E. F.) The plastic esophagus *J Int Coll Surg* 18(5) 695-699 1952
- Brehan (J.) Nouvelle observation d'œsophagoplastie à l'aide du colon droit (Esophagoplasty by use of the right colon A new observation) *Mém Acad chir* Paris 78(10-11) 279-281 1952

- Berman (F F) A plastic prosthesis for resected esophagus *Arch Surg*, 65(6) 916-919, 1952
- Ripley (H R) Olsen (A M) and Kirklin (J W) Esophagitis after esophagogastric anastomosis *Surgery*, 33(1) 1-9 1952
- Ruffo (A) Resezione dell'esofago toracico alto e sostituzione con tubi di materia plastica (Studio sperimentale) (Resection of the upper portion of the thoracic esophagus and substitution of plastic tubes [Experimental study]) *Minerva chir Torino* 7(9) 336-343 1952
- Berman (F F) The experimental replacement of portions of the esophagus by a plastic tube *Ann Surg*, 135(3) and *An Cirurg (B Aires)* 16(3) 337-343 1952
- Chalnot (P), Benichoux (K) and Gille (P) Prothèse de l'œsophage en matière plastique (Documents expérimentaux) Plastic prosthesis for the esophagus [experimental study] *Presse méd*, 60(82) 1742-1744 1952
- Villar (J) and Chavannaz (J) L'œsophagoplastie pré thoracique à l'aide du colon droit et de l'iléon terminal (Ileothoracic esophagoplasty by use of the right colon and the terminal ileum) *Mém Acad chir Paris* 78(20-21) 609-611, 1952
- Miglietta (M) and Noh (G) Plastica esofagea con tubi di politere (Ricerche sperimentali) (Plastic repair of the esophagus with polythene tubes [Experimental investigation]) *Rassual chir med Genova* 2(11) 765-774 1953
- Fry (W) Experimental intrathoracic substitution of the right colon for the resected esophagus *Surg Gynec & Obs* 96(3) 315-322, 1953
- Dop (J) and Dalmás (H) Technique de l'œsophagoplastie préthoracique avec l'iléo-colon en un temps à deux équipes (Technique of a one stage two team prethoracic esophagoplasty with ileo colon) *Asarielle chir* 5(2) 119-134 1953
- Montenegro (F B) Cutait (D) Franganillo (M) Ramos de Oliveira (M) Faria (S G) and Morgante (P A) Transithoracic esophagoplasty for benign stricture by means of the transverse colon *Surgery* 34(2) 313-318 1953
- Dogliotti (A M) and Guglielmini (G) Esofagocolonplastica totale isoperistaltica retrosternale (Total retrosternal esophagoplasty by means of isoperistaltic colon transplantation) *Minerva chir Torino* 8(12) 515-520 1953
- Maccas Un cas d'œsophagoplastie cervicale rétrosternale par le colon droit Succès parfait (Cervical retrosternal esophagoplasty using the ascending colon, with complete success) *Mém Acad chir Paris* 79(21-24) 540-544 1953
- Wawro (N W) Fatal complications after esophageal replacement with plastic tube *Surgery* 36(5) 903-905, 1954
- Mustard (R A) Reconstruction of the esophagus *Surg Clin N Amer* 34 919-995 1954
- Mahoney (C B) and Sherman (C D Jr) Total esophagoplasty using intrathoracic right colon *Surgery* 35(6) 937-946 1954
- Gergely (R) Bedeutung der Mesenteriummobilisierung in der antethorakalen Ösophagoplastik (The value of mobilization of the mesentery in antethoracic esophagoplasty) *Zbl Chir* 79(17) 705-710 1954
- Javid (H) Esophageal reconstruction using colon and terminal ileum *Surgery* 36(1) 132-135 1954
- Heimlich (H J) and Winfield (J M) The use of a gastric tube to replace or bypass the esophagus *Surgery* 37(4) 549-559 1955
- Betts (R H) Thomas (T) and Gopinath (N) Cervical esophagogastric anastomosis for extensive strictures of the esophagus *Surgery* 38(3) 553-565 1955
- Petrov (B A) Reconstructive surgery of the esophagus *Indian J Surg* 1(3) 236-240 1955
- Sherman (C D Jr) Mahoney (C B) Dale (W A) and Stabins (S J) Intrathoracic transplantation of the right colon for esophageal reconstruction *Cancer* 8(6) 1198-1205 1955
- Carwell (J Jr) Hays (R P) and Viar (W N) Notes on conservation of gastric and duodenal function in palliative esophageal reconstruction *Surg* 37(2) 204-212 1955

Chapter 21 : Communications with the Air Passages (Esophagobronchial and Tracheal Fistulae)

- Gut (A) Les fistules œsophago-trachéales *Rev de Laryngol* 47 1921, pp 175 et 237
- Barrut (A) Les fistules œsophago-bronchiques *Thèse de Lyon* no 104 1934-1935
- Monseñat (J) Fistulas tuberculosas esofago-trachéo-bronquicas *Arch de la Soc argent de Anat normal y patol*, Buenos Aires 2 1 à 5 avril août 1940
- Van Nieuwenhuysen Ueber Lungen Tuberculose mit Perforation tuberculöser Drüsen in Bronchi Trachea und Ösophagus *Nederl tijdsch voor geneesk* 1940 pp 4354-4359

- Lempino (G) Tragitto fistoloso tra esofago e fossetta sopraclaveare sinistra *Radiol med* 29 1942 pp 230-232
- Chimani (F) Ueber die erworbenne oesophago tracheale Fistel *Wiener klin Wschr* 1 1943 pp 150-153
- Piquet et Dubuisson Fistule œsophagienne d'origine indéterminée *L Lcho méd du Nord* Lille no 7 juil 1944 p 131
- Langeron Didier, Legrand et Dubuisson Fistule œsophago pleurale *J des Sc méd de Lille* 67 22 29 oct 1944
- Le Lourd Fistule œsophago pleurale chez un tuberculeux pulmonaire *Rev de la tub* Paris 5th série 9 7-9 1944-1945
- Kolman Robertson et Campbell (R T) Un cas de fistule broncho œsophagienne avec absence de fistule pulmonaire *Brit Med J* 12 mai 1945
- Melanec (A) et Zimmerman Fistule œsophago bronchique : diverticule œsophagien *Am J Rontg and Rad* janv 1946
- Clagett (O T) Payne (J H) et Mersch (H J) Acquired esophago tracheo bronchial fistula *Surg Gynec & Obst* 82 janv 1946 pp 87-90
- Sabene Un cas de fistulisation œsophago bronchique par cancer de l'œsophage *Boll d mal d orecchio* janv fév mars 1946 p 4
- Soulas Aboulker et Autier Fistule œsophago pulmonaire dans une sténose cicatricielle de l'œsophage *Ann d oto laryngol* 13 5-6 mai juin 1946
- Brewer (A) Fistule œsophago pleurale complication d'un empyème *Radiology* 47 4 oct 1946
- Mustakallio (S) Fistule œsophago trachéale d'origine lympho granulomateuse *Ann chir et gyn Fennia* 35 2 1946 pp 136-141
- Carrega Casaffousth et Brage (D) Sur un cas de fistule œsophago trachéale d'origine neoplasique à point de départ laryngo trachéal *Semana med* 53 18 1946 pp 789-793
- Adams et Mabley Fistule œsophago trachéale due à une blessure non pénétrante (écrasement) *J Thoracic Surg* 15 4 1946 pp 290-292
- Prouzet et Escande Fistule œsophago trachéale au cours d'un cancer de l'œsophage découverte radiologique *Soc de m'd chir et pharm de Toulouse* mars 1947
- Montserrat (J) Les fistules de l'œsophage d'origine néoplasique *Arch Soc argent Anat normal y patol* Buenos Aires 1 2 mai 1947
- Brewer (Arthur A) Fistule œsophago pleurale complication de pleurésie purulente *Quart Rev O R L* juin 1947
- Mayersohn (M) Copelman (L) et Fenyes (N) Fistule œsophago bronchique *Ann d oto laryngol* oct 1947
- Majer (E H) Fistule de l'œsophage après thyroïdectomie *Soc autrich d oto laryngol* 2 déc 1947 in *Monats fur Ohrenh* mars 1948
- Collao (C E) Fistule œsophago thoracique *Bot de la Soc de cir de Chile* Santiago (Chili) 25 4 déc 1947 pp 224-228
- Stig Berplund An œsophago bronchial anastomosis existing forty years *Acta radiol* 78 3 1947 pp 236-240
- White et Birdsong Fistule trachéo œsophagienne avec rétrécissement *J de chir* 63 12 1947
- Pallard (F) Bérard (M) Planchu (M) Galy (P) et Dumarest (J) Fistule œsophago bronchique basse et abcès du poumon *Soc med d hop de Lyon* 9 fév 1948
- Lowys (P) Tison (I) et Le Barre (H) Mort subite d'un enfant atteint d'adénopathie trachéo bronchique tuberculeuse (rupture d'un ganglion juxta trachéal et fistule broncho œsophagienne) *Soc d et sc sur la tub* 14 fév 1948 C F 1948 pp 381-384
- Ivy (R H) Hawthorne (R H) et Ritter (J A) Reconstruction d'un œsophage en tube cutané après traitement chirurgical d'une fistule trachéo œsophagienne *Plastic and Reconstr Surg* 3 2 mars 1948 p 173
- Bérard (M) et Jurtin Les fistules œsophago pleurales et extra pleurales *Soc d et sc sur la tub* 12 juin 1948
- Testa (C) et Pisani (A) Sur un cas de fistule œsophago gastrique *Minerva med* 39 34 25 aout 1948
- Fontan Verger Moules et Carles Fistule œsophago trachéale chez un nouveau né sans aplasie œsophagienne survie de deux ans *J de méd de Bordeaux et du Sud Ou st* 125 11 aout 1948
- Levrat (M) et Despierrres (G) A propos des fistules œsophago aériennes dans le cancer œsophagien *J méd de Lyon* no 698 5 sept 1948 pp 613-616
- Hughes (W) Fistule œsophago pleurale après perforation de l'œsophage par corps étranger *Am J Dis of Child* Chicago 76 4 oct 1948 pp 401-411

- Auguste (C) Breton (A) et Paris (J) Fistule trachéo-œsophagienne chez une syphilique
Soc de gastro entérol de la Région du Nord 6 fév 1949
- Tulczyński et Zabokrycki (J) Fistule œsophago hépatique *Polski Tygodnik Lekarski*,
Varsovie 4 6 7 fév 1949
- De Iazio (V) Fistule œsophago-épigastrique tuberculeuse *Riforma med* Naples 63 7
fév 1949
- Castells (H R), Bréa (M M) et Lanan (C) (Buenos Aires) Fistule œsophago-bronchique
Rev de la Assoc med argent, 63, 655-656 15-30 juin 1949 pp 259-261
- Albi (R W) et Gilchrist (R K) Fistule œsophago trachéale par traumatisme externe *Arch Surg*, 59 3 sept 1949, pp 454-459
- Munizaguria Minulin et Rapoport Agénès de l'œsophage communication directe trachéo-
œsophagienne *Semana med*, Buenos Aires 56, 41, 13 oct 1949
- Chein (Cl J) Judson (Gl L) et Hemphill (A C) Fistule gastro péricardique consécutive à
une œsophagectomie transthoracique *Ann Surg* 130, 6 dec 1949
- Klarnot Legendre et Gogelot Fistule œsophago extra pleurale dans un double pneumo-
thorax *Rev de la Tub* Paris, 13 7-8 1949
- Soulas et Mounier Kuhn Bronchologie 1 vol, Masson et Cie, édit., 1949
- Arslan (M) Fistule œsophago trachéale traitée par une nouvelle méthode chirurgicale *Acta
di So med chir di Padova* 27, 1, 1949 pp 147-151
- Laforest (A) Contribution à l'étude des fistules œsophago bronchiques *Thèse de Lyon*,
1948-1949
- Charmot Dubuisson (F) Fistules œsophagiennes au cours de la collapsothérapie dans la
tuberculose pulmonaire *Thèse de Paris* 1950, 544
- Terracol (J) et Guérrier (J) Les fausses fistules œsotrachéales *Soc de broncho-œsophro-
gastroc* oct 1950
- Coleman (F P) and Bunch (G H Jr) Acquired nonmalignant esophagotracheobronchial
fistula *J Thoracic Surg* St Louis 19(4) 542-559 1950
- Campi (L) and Grasso (A) Sulle comunicazioni esofago aeree nel corso di neoplasie esofagiche
(Fistulas between the esophagus and the trachea and bronchi in carcinoma of the
esophagus) *Minerva chir* Torino 6(9) 207-212 1951
- De Beaujeu (M I) Fistules œsophago bronchiques chez l'adulte avec diverticule œsophagien
de traction (Esophagobronchial fistula in the adult with traction diverticulum of the
esophagus) *Lyon chir* 47(7) 796-806 1952
- Perronin (J) and Vallée (J) Sixantes cas de carcinome de l'œsophage et de la partie
haute de l'estomac 30 resections et gastrectomies totales (Sixty cases of carcinoma of the
esophagus and of the upper part of the stomach Thirty resections and total gastrectomies)
Presse méd 60(31) 666-669 1952

Chapter 22 Ruptures, Wounds, and Perforations

RUPTURE

- Weder (A) Rupture spontanée de l'œsophage *Acta oto-laryngol* Stockholm 31 5 1943
pp 426-434
- Collis Humphreys et Bond Rupture spontanée de l'œsophage *The Lancet* In *J de chir*,
61 11-12 1945
- Elison (I) et Welty (R) Rupture spontanée de l'œsophage *Surg Gynec & Obst* 83 2
In *J de chir* 63 7-8 1947
- Arbuckle (M F) Rupture traumatique de l'œsophage avec pneumothorax *Ann Otol-
Laryngol* 64 7 1947 p 372
- Frink (N W) Rupture spontanée de l'œsophage *J Thoracic Surg* 16 3 1947 pp 291-297
- De Brux (J) et Busser Sclérose isémique avec anévrisme en aval de la sclérose Rupture de
l'œsophage Aorte brachiale *C R de la Soc anat de Paris* 13 déc 1947 pp 37-40
- Kinsella (T J) Morse (R W) et Hertzog (A J) Rupture spontanée de l'œsophage *J
Thoracic Surg* 17 5 oct 1948 pp 613-631
- Moore (J A) et Murphy (J D) Rupture complète de l'œsophage *J Thoracic Surg* 17 5
oct 1948 pp 632-638
- Higginson (J F) et Clagett (O T) Rupture complète de l'œsophage *J Thoracic Surg*
Saint Louis 17 6 déc 1948 pp 846-848
- Scholefield Perforation spontanée de l'œsophage *Brit Med J* no 4599 2 fév 1949 pp
348-350
- Beal (J M) Rupture spontanée de l'œsophage *Ann Surg* 179 4 avril 1949 pp 512-516
- Fincher (E F) et Swanson (H S) Rupture œsophagienne compliquant une craniotomie

- syndrome complexe Proposition de traitement chirurgical *Ann Surg* 129 5 mai 1949 pp 619-630
- Layne (J) et Hildebrand (E) Rupture de l'œsophage consécutive à un vomissement sévère *Gastro-Enterol* 2 12 août 1949
- Garnier (C M) Rupture spontanée de l'œsophage *Arch Surg* Chicago 59 3 sept. 1949 pp 710-713
- McGregor Gardner Rupture spontanée de l'œsophage *Arch Surg* 58 3 sept 1949
- Cliffon (E F) Rupture spontanée de l'œsophage *Ann Surg* 130 6 déc 1949 pp 1066-1073
- Herrmann (H) La rupture par éclatement de l'œsophage par action interne des ondes de détonation *Zeit fur Laryngol u s w* Stuttgart 79 1 janv 1950
- Kennard (H) La rupture de l'œsophage au cours de l'accouchement *Brit Med J* no 4650 18 fév 1950
- Barrett (N R) Report of a case of spontaneous perforation of the œsophagus successfully treated by operation *Brit J Surg* 35(138) 216-218 1947
- Gardner (C. McG) Spontaneous rupture of the esophagus *Arch Surg* 59(3) 710-713 1949
- Kinsella (T J) Spontaneous perforation of the esophagus *Am Surgeon* 17(7) 584-597 1951
- Anderson (R. L) Rupture of the esophagus *J Thoracic Surg* 24(4) 369-388 1952
- Mackler (S A) Spontaneous rupture of the esophagus An experimental and clinical study *Surg Gynec & Obst* 95(3) 345-356 1952
- Carbonnet and Couinaud Diagnostic des ruptures et perforations spontanées de l'œsophage (Diagnosis of spontaneous ruptures and perforations of the esophagus) *Presse méd* 60(29) 622 1952
- Alt (R. E) Bixby (F E) and Rowell (R J) Spontaneous rupture of the normal esophagus *New England J Med* 749(26) 1060-1062 1953
- Marcozzi (G) and Bellavia (A) Sulle cosiddette rotture e perforazioni spontanee dell'esofago (So-called spontaneous perforation of the esophagus) *Ann ital chir* 32(9) 765-791 1955
- Starkey (G W B) Spontaneous rupture of the esophagus (with presentation of four new cases) *J Thoracic Surg* 30(3) 300-315 1955
- Kirby (C K) Spontaneous rupture of the esophagus *Am J Med Sci* 279(2) 199-204 1955
- Barthel (H) Berstungsruptur des Oesophagus durch Kesselexplosion (Burstung rupture of the esophagus due to explosion of a boiler) *Thoraxchir* (Stuttgart) 2(4) 314-320 1955
- Kerr (H H) Sloan (H) and O'Brien (C E) Rupture of the esophagus by compressed air Case report. *Surgery* 33(3) 417-420 1955

WOUNDS AND PERFORATIONS

- Hetzar Sur les blessures par coup de feu de l'œsophage *Zentralb fur Chir* Leipzig 68 16 19 avril 1941
- Fesh (E J) Plaie par balle de l'œsophage thoracique *Arch Surg* 52 2 fév 1946 pp 172-176
- Knight (J S) Blessures de la trachée et de l'œsophage reçues au combat *The Ann Otol Rhinol and Laryngol* 55 3 sept 1946
- Jiano (J) (rapport Truffert) Plaies de la carotide primitive du nerf pneumogastrique du nerf sympathique du plexus cervical Contusion du plexus brachial Plaies de l'œsophage Ligature de la carotide suture du pneumogastrique et de l'œsophage *Acad de chir* 5 fév 1947
- Davis (J E) Dissection traumatique de l'œsophage médiastinite avec guérison *Arch Oto-Laryngol* 46 2 août 1947 pp 237-239
- Kay (E B) Lésions chirurgicales de l'œsophage observées dans un Centre de Chirurgie thoracique de l'armée *J Thoracic Surg* 16 3 1947 pp 207-214
- Haffett (G St J) Swallowed foreign bodies Some cases treated in a military hospital *Brit Med J* 4497 1947 pp 334-335
- Lyman (E H) A case of esophageal foreign body with mediastinitis and other complications *Laryngoscope* 57 6 1947 pp 400-413
- Sommer Jr (G) et O'Brien (Ch) Blessures de guerre de l'œsophage *J Thoracic Surg*, Saint Louis 17 3 juin 1948
- Wiethe (C) Verätzungen und Verletzungen des Oesophagus *Mischr Ohrenh* Wien 83 4-6 avril juin 1949 pp 196-203

- Matheson (I.) Foreign bodies in the œsophagus, a review of 602 cases *J Lar Otol* Londres 63 8, août 1949 pp 435-445
- Albi (R. W.) et Gilchrist (R. K.) Fistule œsophago trachéale par traumatisme externe *Arch Surg* Chicago 59 3, sept 1949 pp 454-459
- River (L.) Subdiaphragmatic perforation of the œsophagus Importance of jejunal alimentation in delayed surgical treatment *J Int Coll Surg* 14, 5 533-537 1950
- Weisel (W.) and Raine (T.) Surgical treatment of traumatic œsophageal perforation *Surg Gynec & Obst* 94(3) 337-346, 1952
- Barrett (N. R.) *Esophageal Trauma* Modern Trends in Gastro-enterology London Butterworth & Co, 1952
- Samson (P. C.) Injuries and wounds of the œsophagus (A classification) *California Med* 80(5) 363-368 1954

Chapter 23 Instrumental Perforation

- Grace (K.) et Irwin (Th.) La perforation de l'œsophage cervical avec médiastinite *Surgery* 8 6 juin 1940
- Van Grœnendael (M. T.) Perforations de l'œsophage cervical consécutives à la déglutition de corps étrangers *Thèse Paris*, 1940
- Holinger Complications des perforations de l'œsophage *Ann Otol* 1941
- Tourol Perforation de l'œsophage par gastroscopie *Ann Surg*, 114 1941 p 369
- Baum Corps étranger de l'œsophage, perforation médiastinite guérison *Arch oto-rhino-laryngol* 1943
- Hofst (G.) Koch (E.) et Kratschwill Les perforations de l'œsophage *Ann d oto-laryngol* 1943 p 119
- Sacquépée Hache et Baldenweck Cervicotomie pour perforation de l'œsophage *Presse m'd*, no 20 9 déc 1944
- Naudeau Perforation de l'œsophage cervical consécutive à un corps étranger dégluti *Sic d chir de Lyon* no 14 déc 1944
- Barrett (N.) Perforation spontanée de l'œsophage *Thorax* Londres 1 1 mars 1946
- Engler (C. W.) Perforation non instrumentale de l'œsophage *Ann Otol Rhin and Laryng* 55 3 sept 1946
- Klotz (P. L.) Les perforations traumatiques de l'œsophage *Ga d hôp* 119 25 1946 p 509-512
- Kecht (B.) Un cas de lésion fatale de la muqueuse œsophagienne simulant une perforation péritonéale *Acta oto-laryngol*, 34 6 1946 pp 587-592
- Lederer (F. L.) Grossman (A. A.) et Donnelly (W. A.) Conceptions actuelles dans le traitement des perforations œsophagiennes *Ann Otol* 1946
- Moulouguet et Bouche Le traitement des perforations de l'œsophagoscopie *Soc de laryngol d hôp de Paris* 1946
- Frink (N. W.) Perforation spontanée de l'œsophage *J Thoracic Surg* 16 3 juin 1947, pp 291-297
- Heady (C. A.) Perforation de l'extrémité inférieure de l'œsophage Chimiothérapie gastrotomie Guérison *C R de la Soc de laryngol Réunion de l'Assoc amér de Broncho-Œsophagol* in *Ann d O R L* 64 7 juil 1947
- Elly (W. A.) Present day concepts on the treatment of œsophageal perforations *Ann Otol Rhin* 56 4 déc 1947 pp 867-891
- Barrett (N. R.) Un cas de perforation spontanée de l'œsophage traitée avec succès par l'opéra-
tion *Brit J Surg* 35 138 1947 pp 216-218
- Withalm (A.) Laugstenose des Œsophagus Selbstbougerung Perforation Péritonitis nach Gastrostomie *Monatschr f Ohren* 81, 1 1947, pp 417-418
- Culver (G. J.) et Clark (S. B.) Le diagnostic radiographique des perforations du tractus digestif supérieur dans le médiastin et la cavité pleurale *Surgery* 33 3 1947 pp 458-565
- Dorsey (J.) Traitement chirurgical de la perforation de l'œsophage *Arch Surg* 56 1, janv 1948 pp 24-33
- Rebattu et Gaillard Deux cas de rétrécissement cicatriciel de l'œsophage avec accidents graves au cours de la dilatation Guérison par la pénicilline *Soc de laryngol d hôp de Paris* in *Ann d oto-laryngol* 15 mars 1948
- Witherspoon (W.) Un cas d'estomac en verre de montre gastroscopie perforation par sondes phagie guérison *Gastro-Enterol* 10 3 mars 1948
- Baumann Deux cas de péritonite par perforation de l'œsophage abdominal par sondes dilatatrices *Acad de chir* 14 avril 1948 in *Mém Acad* 74 11-12 pp 307-304

- Moulouguet (A) et Bouche (J) Le traitement des perforations de l'œsophage A propos de 3 cas guéris *Ann d oto laryngol*, 65 5-6 1948 mai juin 1948
- Golcher (J) Perforation pharyngo œsophagienne post cricoldienne due à une endoscopie traitée par la suture immédiate *Lancet* 254, 6513 26 juin 1948
- Guns Perforations œsophagiennes et pénicilline *XLVI Congrès franç d Oto Rhino Laryng* 18-23 oct 1948
- Escat (M) Perforations de l'œsophage abdominal au cours des cathétérismes dilateurs à la bougie *XLVI Congrès franç d O R L* Paris 18-23 oct 1948
- Jemern (E) Les résultats du traitement de la perforation de l'œsophage *Ann Surg* 128 5 nov 1948
- Lindert (M C F) et Winnik (D) Perforation of the aorta a complication of carcinoma of the esophagus *Am J Digest Dis*, 15 11 nov 1948 pp 378-381
- Tailens (J P) Perforation œsophagienne par corps étrangers et traitement d'urgence *Practica oto-rhino laryngol* 10 1-2 1948
- Terracol (J) Guerrier (Y) et Chalmeton (J) Perforations de l'œsophage par auto cathétérisme importance de la contracture abdominale *Oto Rhino Laryngol internat* no 10 1948
- Collins (E G) Cardiospasm traumatic perforation of œsophagus empyema left chest drainage of empyema gastrostomy recovery *J Laryng & Otol* 62 1948 pp 709-711
- Watson Williams (E) Post cricoid pharyngo œsophageal perforation *Lancet* 2 1948 p 34
- Resano (T H) Le traitement chirurgical des perforations de l'œsophage *Rev de chir* 68 1-2 janv fév 1949 pp 1-11
- Scholefield (J) Perforation spontanée de l'œsophage *Brit Med J* 4599 26 fev 1949 pp 548-550
- Perrotin et Frileux (Rapport de M. Quénu) Perforation traumatique de l'œsophage inférieur Thoracotomie d'urgence Suture Guérison *Mém de l'Acad de chir* 75 9-10 mars 1949 pp 237-247
- Blondin (M S) A propos des perforations traumatiques de l'œsophage à la suite de manœuvres endoscopiques *Acad de chir* 23 mars 1949
- Asher (L) et Sidney Cohen Perforation de l'œsophage et de l'estomac dans la gastroscopie 3 observations *Gastro Entérol* Baltimore 17 6 juin 1949
- Bigard (J D) et Kerr (H) Le traitement chirurgical de la perforation instrumentale de l'œsophage *Arch Surg* 58 6 juin 1949
- Wells (C) Hughes (J H) Hedwards (R) et Marcus (R) Perforations consecutives à la dilatation des strictures œsophagiennes *Thorax* Londres 4 2 juin 1949
- Rea (R P) Perforation de l'œsophage par une pointe fixatrice ablation par électro aimant *Arch Oto Laryngol* 50 2 aout 1949
- Lynch (J P) Perforation spontanée de l'œsophage *New England J Med* Boston 74 11 15 sept 1949 pp 395-400
- Bruzzone (P L) et Bellion (B) Considerazioni clinico radiologiche sul valore del segno di Minnigerode *Ann di laryng* 48 1949 pp 11-20
- Theikas et Ludin Jr Un cas de perforation de l'œsophage avec médiastinite consecutive *Radiol clin Bale* 19 1 janv fév 1950
- Bigard (J D) and Kerr (H H) Surgical management of instrumental perforation of the esophagus *Arch Surg* 58(6) 739-751 1949
- Kernan (J D) Perforation of the esophagus as a surgical emergency *Surg Clin N Amer* 30 405-419 1950
- Seybold (W D) Johnson (M A) and Leary (W V) Perforation of the esophagus an analysis of 50 cases and an account of experimental studies *Surg Clin N Amer* 30 1155-1183 1950
- Cropper (C F J) Houghton (P) and Kidd (P) Low œsophageal perforation at gastroscopy *Brit Med J* no 4688 1950 p 1099
- Howard (J T) The management of instrumental perforation of the esophagus *Am J Gastroent* 23(5) 399-410 1955
- Palmer (E D) and Wirts (C W) Survey of gastroscopic and esophagoscopic accidents *J A M A* 164(18) 2012-2015 1957

Chapter 24 Foreign Bodies within the Esophagus

Chapter 25 Extraction of Foreign Bodies

- Klicpera (P) Blessure de l'œsophage par corps étranger guérison *Soc tien d O R L* 5 déc 1939

- Danielevitz (J) Les hémorragies œsophagiennes pouvant présenter des difficultés de diagnostic et consécutives à des corps étrangers *N nat's fur Ohrenh und Laryng* - Rhin 1 1939, fasc. 2
- Matus (E I) L'extraction d'un corps étranger sphérique *Brinchosc, Oesophagosc et Gastrosc*, 1939
- Banyai Symptomatology des corps étrangers œsophagiens *Deutsch med Wchenschr* Berlin 65, 46 15 nov 1940
- Guida (Guido) Observations étiologiques cliniques et thérapeutiques sur quelques cas de corps étrangers de l'hypopharynx et de l'œsophage Progrès de technique œsophagoscopique *Il Palsal* a 18 10 oct 1942 19 9 sept 1943
- Huizinga (L) Sur la symptomatologie et le traitement des corps étrangers perforants de l'œsophage *Acta oto-laryngol* Stockholm, 31 1942 pp 279-290
- Pallestrini et Ughetto Syndrome asmatica da corpo estraneo nell'esofago *Liguria med* Luglio, 1942
- Chavanne (L) A propos de six cas de corps étrangers de l'œsophage *I O R L internat* no 9 sept 1944 pp 229-232
- Fisher (C E) Foreign bodies in esophagus and lower respiratory tract *Alabama State Med Assoc J* 13 1944 p 220
- Buzoianu Technique œsophagoscopique pour extraction de prothèse dentaire *Acta oto-laryngol* 32, 1944
- Ombredanne Epingle de sûreté dans l'œsophage d'un nourrisson extraction endoscopique après version dans l'estomac *Arch fran, de pédi* 2, 1 1944 p 123
- Barrie (H) et Townrow Cas de perforation de l'aorte par un corps étranger de l'œsophage *J Laryng et Otol* janv 1946
- Schuermans Obstruction complète de l'œsophage par un noyau de cerise Réunion commune de la Soc belge d'O R L avec la Soc belge de pédi 31 mars 1946 in *O R L intern*, no 4 avril 1947
- Rasquin (P) Corps étranger méconnu du carrefour aéro-gigestif C ngrès belge d'O R L 30 juin 1946
- Granger Quelques cas de corps étrangers des bronches et de l'œsophage *I Afrique fran chir* Alger 4 4 juil août 1946
- Robertson (H) et Fletcher (G) Corps étranger de l'œsophage *Canad Med Assoc J* 3 sept 1946
- Iverett (R) Opération externe pour extraire des corps étrangers dans l'œsophage *Arch ital di otol* nov 1946
- Leto Corps étranger rare de l'œsophage chez un enfant de 3 ans *Arch ital di otol* nov 1946
- Holmes (L M) Corps étranger de l'œsophage *Arch Oto-Laryngol* 44 5 nov 1946
- Ballivet (M) Abscès cervical rétro-œsophagien par corps étranger Pénicilline cervicotomie et aspiration continue *Mém Acad chir* Paris 72 29-30-31 1946 pp 487-491
- Jones (H) Aimant pour l'extraction des corps étrangers dans l'œsophage *Ann oto-laryng* 13 9-10 1946 pp 466-467
- Holinger (P H) Magnets for the extraction of foreign bodies from the air and food passages *Ann Otol Rhin and Laryngol* 55 3 1946 pp 601-603
- Andrescu Braila (C) Consideratiuni asupra corpurilor straini tracheo-broncho-esofagieni la sugari *Rev romana de O R L si broncho-esof*, 13 3-1 1946
- Tavoillot (L) Contribution au traitement des corps étrangers enclavés dans l'œsophage thoracique Leur extraction par thoracotomie transpleurale Thèse Strasbourg 1946-1947
- Sellor (Holmes) Lame de rasoir dans l'œsophage depuis 15 ans ablation par respiration *Surg* 34 135 1947 pp 276-277
- Turner (Grev) Plaque dentaire enclavée dans l'œsophage Bristol 34 135 janv 1947
- Wolcott (Ch) Un cas mortel par corps étrangers œsophagien *Austral State J Med* 47 3 1 fév 1947
- Hennessey (R) Les corps étrangers de l'œsophage *Med J Austral* Sydney 34 19 10 mai 1947
- Lemarié (A) Le problème de l'épingle double en endoscopie œsophagienne *Ann d'otolaryng* 1 64 7 juil 1947
- Roby et Lafourcade Oesophagotomie externe pour enclavement œsophagien d'un dentier au 5 jour *Miro med* Casablanca 76 269 sept 1947 pp 341-347
- Garderes et Ferron Corps étranger de l'œsophage inextirpable par les voies naturelles Infection gangreneuse Oesophagotomie sans sutures profondes Suture cutanée presque complète Pémallinothérapie Guérison *O R L Internat* déc 1947 p 180

- Michăescu (Paunel) et Nicolescu (G.) Corps étranger œsophagien volumineux avec péricœsophagite suppurée et abcès pulmonaire consécutif *Revue roumaine d'O R L* 14 1-4 janv.-déc. 1947 p. 49
- Rosefield (Cl.) Corps étranger enclavé dans l'orifice supérieur de l'œsophage pendant plus de cinq semaines *Med J Austral* 33 24 14 déc. 1946 In *J de chir* 63 6 1947
- Mc Chester (H. L.) Deux cas de médiastinite consécutive à des corps étrangers de l'œsophage *Ann d'oto-laryngol* 1947 p. 380
- Lemarey (A.) Le problème des fragments de verre dans les voies aériennes et l'œsophage *Ann d'oto-laryngol* 1947, p. 553
- Koschier (R.) Fremdkörper im Oesophagus *Wschr Ohrenh* 81 1947 p. 482
- Garshin (M. I.) Aorta hæmorrhage caused by a foreign body of the œsophagus *Vestnik Oto-Rino-Laryngol* no 2 1947 pp. 62-65
- Hallett (G. et J.) Swallowed foreign bodies. Some cases treated in a military Hospital *Brit Med J* 4497, 1947 pp. 334-335
- Hagens (E. W.) Endoscopic use of the biplane fluoroscope *Arch Oto Lar* 46 3 1947 pp. 382-385
- Equen (Alurdock) Magnetic removal of foreign bodies from the food and air passages *Arch Oto-Laryngol* 45 1947 pp. 225-229
- Lell (W. A.) Epingle de sûreté dans l'œsophage avec perforation du péricarde *Ann Oto-Laryngol* 64 7 1947 p. 393
- Silber (S.) et Epstein (B. ■) L'ablation pérorale de certains corps étrangers sans endoscopie (nouveau projet d'utilisation d'un aimant alnico) *New York State J Med* 47 10 1947 pp. 1122-1124
- Schwartz (V. J.) Cas unique de corps étranger de l'œsophage *Arch Oto Laryngol* 45 5 1947 pp. 562-567
- Utrata (J.) Sur la clinique et le traitement des corps étrangers de l'œsophage *Wien med Wochenschr* 98 5-6 7 fév. 1948 pp. 56-60
- Massias (Ch.) et Nguyen Dinh Hao Péricardites putrides 1 Suite d'abcès sous-phrénique par ascarirose intra hépatique 2 Suite d'œsophagite gangréneuse par arrêt de poisson. *Soc méd des hôp de Paris* 7 mai 1948
- Despons (M. J.) Statistique de corps étrangers des voies aéro-digestives *Soc Broncho-Oesophagose et de gastrosc de Langue franç* 8 mai 1948
- Fontaine (R.) Heumendinger (E.) et Warter (P.) A propos d'un corps étranger de l'œsophage (prothèse dentaire en vulcanite) extrait avec succès au bout de deux mois par voie trans thoracique droite *Rev. de chir* Paris 67 5-6 mai juin 1948 pp. 184-192
- Richardson (J. R.) La déglutition des deniers problème œsophagien *Arch Oto-Laryngol* 47 6 juin 1948
- Terracol et Guernier Perforation de l'œsophage par corps étranger *Sem des hôp* no 54 18 juil. 1948
- Hartz (P.) et Ary van der Sar Ulcération aigue de l'œsophage associée des corps intracuticulaires inclus *Gastro Entérol* 10 3 sept. 1948
- Dunif (M. F.) Volumineux corps étranger de l'œsophage un fragment de ceinture de camisole de force *Soc de broncho-œsophagose et de gastrosc de Langue franç* 21 oct. 1948
- Dunif (M. F.) Procédé simple d'extraction chez les bébés des épingles de nourrice ouvertes pointe en haut dans l'hypopharynx *Soc de broncho-œsophagose et gastrosc de Langue franç* 21 oct. 1948
- Pouget (R. J.) Un cas peu banal d'épingle double ouverte intra œsophagienne *Soc de broncho-œsophagose et de gastrosc de Langue franç* 21 oct. 1948
- Joubin (J.) Les corps étrangers de l'œsophage *Progres méd* 76 21 10 nov. 1948 pp. 526-531
- Scheffler Ablation des corps étrangers de l'œsophage *Deutsch med Wochenschr* Stuttgart 73 43-44 19 nov. 1948
- Loubat Fontan Despons Carles et Bouet Corps étranger de l'œsophage extraction chirurgicale *Arch franç de pédi* 5 4 1948
- Von Oppkofer (E. K.) Verblutung aus der Arteria thyroidea inferior nach Speiseröhrenfremdkörper *Practica O R L* 10 1-2 1948
- Corelli (G.) Corps étrangers de l'œsophage difficiles à extraire *Bol d malati d orecchio* Florence 66 3-4 1948 pp. 116-119
- Efskind (L.) Un cas de corps étranger de l'œsophage *Nordisk med* Stockholm 37 6 1948 pp. 288-289
- Bloch (André) et Autier (Ch.) Rétrécissement de l'œsophage par corps étranger méconnu *Bull Soc de broncho-œsophagose et de gastrosc de Langue franç* 1948
- Gatch (W. D.) et Molt (W. F.) Ablation transgastrique des corps étrangers volumineux bloqués dans la partie inférieure de l'œsophage *Arch Surg* 56 1 1948 pp. 21-23

- Lugli (G) Sur le traitement chirurgical et les complications de quelques cas de corps étrangers de l'œsophage *Ann d oto laryngol* 66 1-2, janv fév 1949
- Chavanne (L) Un cas de corps étranger de l'œsophage méconnu à l'examen radioscopique cachet et bouillies opaques *L O R L internat*, 33 2 fév 1949 pp 25-26
- Chevalier Jackson et Chevalier L Jackson Vertebrated magnets for removal of foreign bodies from the air and food passages *Ann Otol Rhinol and Laryngol* mars 1949
- Huizinga (E) Les corps étrangers perforants de l'œsophage *Nederl tijdschr voor geneesk* Haarlem 93 16, 16 avril 1949 pp 1234-1240
- Autier (Ch) A propos de l'extraction des épingles ouvertes en haut dans l'œsophage *Soc de Laryngol des Hop de Paris* 16 mai 1949, in *Bronchosc Gastrosc et Œsophagosc* 1949
- Chevalier L Jackson Nouvel aimant porté sur tige souple pour extraction des corps étrangers *Soc broncho œsophagosc et de gastrosc de Langue franç* 10 juil 1949
- Dumont (P) 1 Curieux voyage d'un corps étranger dans les voies digestives 2 Corps étranger volumineux de l'œsophage *Soc de bronch œsophagosc et de gastrosc de Langue franç*, 10 juil 1949
- Jemerin (E E) et Aronoff (J S) Corps étranger dans la thyroïde suite d'une opération de l'œsophage *Surgery* 25, 1 1949, pp 52-59
- Vesselle (P) Trois observations de corps étrangers multiples des voies aériennes et de l'œsophage *Soc de broncho œsophagosc et de gastrosc de Langue franç* 10 juillet 1949
- Tonndorf Syncope respiratoire mortelle dans le cas d'extraction de corps étrangers de l'œsophage H N O Berlin 1949 I, 11 486-488
- Casanova (M A) Mobilisation de corps étrangers de l'œsophage avec spasmolytiques II *Congrès internat d Oto Laryngol* Londres 18-23 juil 1949
- Tezel (L B) Un cas d'une pièce de monnaie dans l'œsophage *IV Congrès internat d Oto Laryngol* Londres 18-23 juil 1949
- Holinger (P H) Johnston (K C) et Greengard (J) Anomalies congénitales de l'œsophage dénoncées par des corps étrangers œsophagiens *Am J Dis of Child*, vol 78 no 4 oct 1949
- Terracol (J) Gros (Cl) Ybanez et Guerrier (Y) Spondylo arthrite cervicale avec compression médullaire par corps étranger de l'œsophage *Soc des Sc méd du Languedoc* 9 déc 1949
- Goldestein (M A) Zur Kasuistik des Blutflusses aus Speiseröhre und Aorta infolge eines Fremdkörpers der Speiseröhre *Vestn otol i t d* 2, 1949, pp 78-80
- Fabritius (T) Corps étrangers de l'œsophage avec symptômes uniquement respiratoires *Nordisk med Stockholm* 41 19 1949 p 866
- Chiara (G) Corps étranger (épingle de sûreté) retenu dans l'œsophage depuis plus d'un an extraction guérison *Bollet e mem d Soc Tosco Umbra di chir*, Florence 10 3 1949
- Kindler (W) Verlorene künstliche Gebisse als eingebildete Speiseröhrenfremd Körper und deren Verlauf *H N O Berlin*, 1 1949 p 281
- Matheson (J) Corps étrangers de l'œsophage une revue de 602 cas *J Laryngol and Otol* London 63 8 août 1949 pp 435-445
- Dumont (P H) Corps étrangers multiples de la trachée et du tube digestif *Bronchosc et gastrosc*, Paris 1949
- Lebreton Corps étrangers œsophagiens formant soupape *J de radiol, d électrol et Arch d électric méd* Paris 30 11-22 1949
- Van de Calsoy de (P) Corps étrangers méconnus de l'œsophage *LI Congrès ann de la Soc belge d O R L* 25 juin 1950
- Agazzi (C) Il valore del trattamento antibiotico negli interventi di esofagotomia e mediastinotomia per l'estrazione di corpi estranei (The value of treatment with antibiotics in esophagotomy and mediastinotomy for the removal of foreign bodies) *L'alfa* 27(6) 359-376 1951
- Malenchini (M) Resano (H J) and Gugliatella (H) Contribucion al estudio de las complicaciones respiratorias producidas por cuerpos extraños de esófago (Contribution to the study of respiratory complications caused by foreign bodies in the esophagus Case report) *Presse méd argent* 40(49) 3303-3308 1953
- Jackson (C L.) Foreign bodies in the esophagus *Am J Surg*, 93(2) 308-312 1957
- EXTRACTION OF METALLIC FOREIGN BODIES BY JOINTED MAGNETS
- Jackson (Chevalier) Corps étrangers dans la trachée les bronches et l'œsophage L aide apportée dans leur extraction par l'œsophagoscopie la bronchoscopie et le magnétisme *Laryngoscope* 15 avril 1905 pp 257-281
- Jackson (Chevalier) Peroral Endoscopy and Laryngeal Surgery *Laryngoscope* 1915

- Jackson (Chevalier) Aide magnétique dans la bronchoscopie et l'œsophagoscopie Translation des corps étrangers introduits dans les voies respiratoires et de la nutrition *Ann Oto Rhin Laryngol* 37 juin 1928 p 435 et *Proc Am Bronchosc Soc* 1927
- Jackson (Chevalier) et Jackson (Chevalier L.) *Maladies des voies respiratoires et de la nutrition du s a des corps étrangers* W B Saunders Co Philadelphie 1936
- Silber (S) Kaplan (C) et Epstein (B S) L'emploi de l'aimant permanent (Alnico) dans l'extraction d'un corps métallique par la bouche (corps situé dans l'estomac) *Ann Otol Rhinol et Laryngol* 53 sept 1944 p 589
- Equen (Murdoch S) A New Magnet for Removal of Foreign Bodies in Food and Air Passages *JAMA*, 127 13 janv 1945 p 87
- Equen (Murdoch S) Magnetic Removal of Foreign Bodies from Food and Air Passages under Fluoroscopic Guidance *South Med J* 38 1945 p 245
- Holinger (P) Aimants pour l'extraction des corps étrangers hors des voies respiratoires et nutritives *Trans Am Broncho Esophagol Assoc* 1946
- Tucker (G) A Flexible Gastroscopic Forceps with Magnetic Tip *Trans Am Broncho Esophagol Assoc* 1946 p 17
- Equen (Murdoch S) Foreign Bodies in Duodenum *South Med J* 41 mars 1948 p 187
- Equen (Murdoch S) Influence magnétique en bronchoscopie *Laryngoscope* 41 mars 1948 p 187
- Penta (A Q) Electro Magnet for the Removal of Magnetic Objects from the Bronchial and Esophageal Gastric Tract *Trans Am Broncho Esophageal Assoc* 1948 pp 15-16
- Rea (R. P) Perforation de l'œsophage par une punaise métallique Opération externe Extraction avec aimant *Arch Oto Laryngology* no 2 aout 1949

Chapter 26 Benign Tumors

- Haensch (F) Tumeur polypoïde de l'œsophage *Fortschr a Geb d Röntgenstr* 58 3 sept 1938 pp 233-239
- Samson (P C) et Zelman (J) Tumeurs pédiculées de l'œsophage *Arch Oto Laryngol* aout 1942 ■ 203
- Harrington et Moersch Traitement chirurgical des tumeurs bénignes de l'œsophage *J Thoracic Surg* 13 1944 p 934
- Harper et Tivencio Tumeurs bénignes de l'œsophage *Brit J Radio* 18 avril 1945 pp 99-107
- Adams (R) et Hoover (W B) Benign tumors of the esophagus *J Thoracic Surg* 14 aout 1946 p 279
- Calmenson (M) et Clagett (O Th) L'ablation chirurgicale des léiomyomes de l'œsophage *Am J Surg* 72 5 nov 1946
- Bozzi (E) et Cova (P L) Tumeurs polypoides de l'intestin Considerations cliniques anatomiques radiologiques et thérapeutiques *Radiol Med* 33 4 avril 1947 pp 161-208
- Chaffer (P W) et Kittle (C F) Léiomyome œsophagien un cas d'ablation chirurgicale réussie *JAMA* 133 16 19 avril 1947 pp 1202-1205
- Guiffrida (E) Angiome de l'œsophage Les tumeurs bénignes de l'œsophage *Minerva med* Turin 38 31 4 aout 1947
- Mc Peak et Arons (W) Adénocanthome de l'œsophage (un cas) ressemblance de la tumeur avec la tumeur de la glande salivaire *Arch Pathol* 41 4 oct 1947
- Fanew (N) Contribution au diagnostic des tumeurs polypeuses de l'œsophage *Wien klin Wochenschr* 59 45 14 nov 1947
- Jones (J C) Dédoubléments œsophagiens en kystes médiastinaux d'origine intestinale *J de l'Ouest de chir obst et gyn* 55 11 1947 pp 610-617
- Moersch (H J) et Harrington (S W) Tumeurs bénignes de l'œsophage *Ann d oto laryngol* 1947 p 394
- Tucker (G) Kyste congénital du médiastin produisant une compression de la partie inférieure de la trachée et de l'œsophage *Ann d oto laryngol* 1947 p 597
- Ferguson (Ch F) et Hackworth (L E) Hemangioma of the esophagus in infancy *Arch Oto Laryngol* 45 5 1947 pp 585-592
- Lanzara (Antonio) Léiomyome de l'œsophage et syndrome de dysphagie fonctionnelle *Arch de chir del Torace* ? 1947, pp 283-292
- Menod (O) Kyste de l'œsophage simulant un neurinome diagnostic histologique après ablation *Acad de chir* 1947
- Mikhelovich (M S) Polypes de l'œsophage *Am Rev Sov Med* New York ■ avril 1948 pp 97-129

- Kenworthy (R.) et Welch (C. S.) Léiomyomes de l'œsophage et du cardia gastrique *Surgery* 23 5 mai 1948 pp 745-752
- Tanew (N.) Tumeur œsophagienne *Sem d s hôp* no 42 2 juin 1948
- Bozzi (E.) et Cova P. L.) Sur les tumeurs polypeuses de l'œsophage *Ann d'oto-laryn ol* 65, 7 juil 1948 p 433
- Bello (M.) Polype de l'œsophage *Prensa med argent* Buenos Aires 35 42 15 oct 1948 pp 1987-1992
- Beegler (R.) Collins (J.) et Hall (M.) Les tumeurs œsophagiennes pédiculées bénignes *Am J Surg* 60 4 oct 1948 pp 466-470
- Bordone Léiomyome de l'œsophage Etude clinique et anatomique *Arch ital di otol* Milan 59 7 1948, pp 571-581
- Lortat Jacob (J. L.) Deux léiomyomes de l'œsophage *Sem des hôp d Paris* 30 mai 1949
- Harrington (S. W.) Traitement chirurgical des tumeurs bénignes ou secondairement malignes de l'œsophage *Arch Surg*, Chicago 58 5 mai 1949 pp 646-661
- Leroux (L.) Les tumeurs bénignes de l'œsophage *Bronchosc œsophage et gastrosce* Paris an. 1949
- Chu (P. S. H.) et Adams (W. E.) Tumeurs bénignes de l'œsophage un cas de léiomyome *Arch Surg*, 60 1 janv 1950 pp 92-101
- Cornell (A.) et coll Cryst of the esophagus *Gastroenterology* 15 2 1950 pp 260-263
- Lortat Jacob (L. J.) Peut-on envisager le démembrement des syndromes fonctionnels de l'œsophage? La myomatose diffuse de l'œsophage (Entité anatomo-clinique non encore décrite) *Sem des hôp* Paris, 36, 5, janv 1950
- Goldmann (A.) et Masters (H.) Léiomyomes de l'œsophage *Arch Surg* 60 mars 1950 pp 559-574
- Lortat Jacob (J. L.) Myomatoses localisées et myomatoses diffuses de l'œsophage *Arch d mal de l'appareil dig*, 39 4 avril 1950
- Thomeret Kyste de l'œsophage — Jung Léiomyome de l'œsophage (rapport Patel) *Acad de chir*, 24 mai 1950
- Nishikawa (H.) Pharyngo-œsophageal polyp *Otolaryngol* 30, Nippon Igaku Zasshi 44 21(2) 31-34, 1949
- Cornell (A.), Blumberg (M. L.) and Sarot (I. A.) Cryst of the esophagus Case report and review of literature *Gastroenterology* 15(2) 260-263 1950
- Gledhill (E. Y.) and Morrow (A. G.) Calciated epithelial cyst of the esophagus Report of a case *J Thoracic Surg* 22(6) 923-927 1950
- Cornell (N. W.) Shehadi (W. H.) and Sharnoff (J. G.) Leiomyomas of the esophagus report of five cases *Surgery* 28(5) 881-886 1950
- Palanker (H. K.) Constantine (A. B.) and Paine (J. R.) Successful resection of an angioend-thelioma of the cervical esophagus Case report and review of the literature *Arch Surg* 67(5) 627-633 1951
- Myers (R. T.) and Bradshaw (H. H.) Benign intramural tumors and cysts of the esophagus *J Thoracic Surg* 21(5) 470-482 1951
- Chalnot Faivre Lochard and Hahn Les léiomyomes de l'œsophage et leur traitement par énucléation simple (Leiomyomas of the esophagus and their treatment by simple enucleation) *Presse med* 59(12) 224-226, 1951
- Lortat Jacob (J. L.) La myomatose nodulaire diffuse de l'œsophage (Diffuse nodular myomatosis of the esophagus) *Ga- méd portug* 5(1) 57-64 1952
- Johnston (J. B.) Clagett (O. T.) and McDonald (J. R.) Smooth muscle tumors of the esophagus *Thorax* 8(4) 251-265 1953
- Viereck (H. J.) Zirkulär wachsendes Myom des Ösophagus (Circular myoma of the esophagus) *Thorax Chir* (Stuttgart) 1(4) 309-315 1953
- Totten (R. S.) Stout (A. P.) Humphreys (G. H.) and Moore (R. L.) Benign tumors and cysts of the esophagus *J Thoracic Surg* 25(6) 606-622, 1953
- Baumann (W.) and Von Hasselbach (H.) Ein Beitrag zur Erkennung und Behandlung der gutartigen gestielten Tumoren des oberen Ösophagus (Contribution to the diagnosis and treatment of benign pedicled tumors of the proximal esophagus) *Arch klin Chir*, 274(5) 347-352 1953
- Krauss (H.) and Hutschenreuter (W.) Diagnose und Therapie gutartiger Tumoren der thorakalen Speiseröhre (Diagnosis and treatment of benign tumors of the thoracic esophagus) *Dtsch med Wschr* 79(8) 297-299 1954
- Creech (O. Jr.) and De Bakey (M. E.) Calciated epithelial cyst of the esophagus associated with cardiac abnormalities Report of two cases *J Thoracic Surg* 28 1) 64-77 1954

- Mousseau (M) Hardy (M) and Kerneis (J P) Myomatose diffuse de l'œsophage (Diffuse myomatosis of the esophagus) *Arch mal appar dig* 43(11) 1071-1075 1954
- Callanan (J G) Simultaneous occurrence of simple and malignant tumors in the esophagus *J Thoracic Surg* 28(1) 4-10 1954
- Sweet (R H) Soutter (L) and Tejada Valenzuela (C) Muscle wall tumors of the esophagus *J Thoracic Surg*, 27(1) 13-35 1954
- Di Matteo (G) Resezione esofagocardiogastrica per leiomioma gigante. Considerazioni anatomo-cliniche e chirurgiche sui tumori muscolari dell'esofago (Resection of esophagus cardia and stomach for giant leiomyoma. Anatomical clinical and surgical aspects of muscle tumors of the esophagus) *Arch ital chir* 79(5) 352-370 1955

Chapter 27 Malignant Tumors

- Resch (B) Ein Fall von Karzinom des Oesophagus *Zbl fur allg Path* 53 1931-1932 pp 235-238
- Mathias (H G) Cancer de l'œsophage avec dilatation idiopathique de l'œsophage *Roentgen praxis* 10 2 fév 1938 pp 107-110
- Cain Cattani and Sikorav La perforation du cancer de l'œsophage dans la trachée et dans les bronches *Arch mal app dig* 28 8 oct 1938
- Proske (E) Recherches sur les métastases de l'œsophage *Strahlentherapie* 64 2 1 fév 1939 pp 227-248
- Finger Les tumeurs de l'œsophage *Munchen med Wochenschr* 86 46 17 nov 1939
- Benedict (E H) Carcinoma of the esophagus with special reference to treatment by radical surgery, X ray and bouginage *Ann Otol Rhin and Laryng* 50 déc 1941 pp 1249-1263
- Brulé (M) Hillemand (P) et Audoly (P) Cancer de l'œsophage avec métastase dans le muscle cardiaque *Arch d mal de l'app dig* 30 1941
- French et Garland Léiomyosarcome de l'œsophage *Am J Roent* 45 1941 pp 27-31
- Adams (H D) Carcinoma of the esophagus *Surg Clin North Am* 22 juin 1942 pp 703-719
- Samson (P C) et Zelman (J) Tumeurs pédiculées de l'œsophage *Arch Oto Laryng* 36 1 août 1942 p 203
- Pennes (A E) Léiomyosarcome de l'œsophage *Am J Roent* 48 sept 1942 pp 336-340
- Liaras (H) Cancer du cardia et méga œsophage *Soc de chir d Alger* 20 nov 1942 pp 6-7-8-9
- Moure et Prat Rousseau Deux cas de néoplasme de l'œsophage à symptômes anormaux *J de med de Bordeaux et du Sud Ouest* 171 1-2 15-30 janv 1944 p 130
- Mayoux (R) et Martin (H) Epithélioma développé sur méga œsophage *Ann d oto laryngol* nos 4-5-6 avril mai juin 1944 p 86
- Santy (P) et Bérard (M) Les tumeurs intra thoraciques dites du médiastin postérieur *Lyon chir* no 6 nov déc 1944 pp 121-122
- Buschke (F) et Cantini (S T) Supervoltage Roentgen therapy of esophageal carcinoma *Radiology* 42 1944 p 480
- Wasch (M G) et Epstein (B S) Roentgen visualization of tumor of cardia *Am J Roent and Rad Ther* 111 51 1944 p 572
- Mayoux et Dumarest Sténose œsophagienne d'aspect cicatriciel de nature cancéreuse métastatique *Soc d O R L Lyon* 11 12 fév 1945 p 336
- Churay Debray et Choubrac Fistule œsophago-pulmonaire de type rare au cours d'un cancer de l'œsophage *Gaz méd de France Paris* 53 5 1 mars 1946 In *J de chir* 63 3 1947
- Eysauiet Paralysies récurrentelles doubles du cancer de l'œsophage *Soc O R L de Lyon* 14 avril 1946 in *Lyon méd* 11 1947 p 453
- Tanner (N) Le carcinome de l'œsophage terminal et du cardia *Proc Roy Soc of Med* Londres 39 7 mai 1946
- Guzzetti (J C) Le cancer de l'œsophage et son traitement *Bot de Sanat Sao Lucas Sao Paulo* 8 11 mai 1946 (suppl de *Anais Paulist de med e cir*)
- Resano (J H) Commentaires sur le diagnostic et le thérapeutique du cancer de l'œsophage *Prensa med argens* 33 18 mai 1946 pp 912 à 928
- Rebattu Rabaste et Lancon Cancer de l'œsophage sans signes radioscopiques - décelé par l'œsophagoscopie *Lyon méd* 78 176/23 9 juin 1946
- Bensaude (A) et Vigue (R) Mégaœsophage et cancer *Soc de gastro entérol de Paris* 9 déc 1946
- Jacobson (F) Un cas de cancer de l'œsophage vivant et libre de toute tumeur six ans après un traitement radiothérapique *Acta radiol* 27 3-4 1946 pp 351-357
- Fagan (A P) Un moyen de diagnostic précoce du cancer de l'œsophage *Irish M J Sc* 6 242 1946 pp 51-55

- Resano (H J) Nouvelles considérations sur l'œsophagoscopie et la biopsie dans le cancer de l'œsophage *Rev argent O R L*, Buenos Aires 15 3-4 1946 pp 94-105
- Carrega (N) Pronostic du cancer œsophagien *Sud méd et chir* 78 2277, 1946 pp 109-111
- Postoloff (A V) et Cannon (W M) Genèse d'une perforation aortique secondaire à un cancer de l'œsophage Rapport d'observation dans deux cas *Arch Pathol* 41, 5, 1946 pp 533-539
- Tanner (N C) Present position of cancer of the esophagus *Post Grad M J* 23 mars 1947, pp 109-139
- Rebattu, Rabaste et Martin Syndrome de Tapia au cours d'un cancer de l'œsophage *Soc O R-L de Lyon*, 10 fév 1947 in *Lyon méd* 1, 1948, p 66
- Rebattu, Martin et Gaillard Cancer de l'œsophage rapidement métastatique *Soc O R-L de Lyon*, 10 fév 1947 in *Lyon méd* 1, 1948, p 67
- Darin Pupil (I) Le diagnostic précoce dans la résection radicale du carcinome de l'œsophage inférieur *Am J Surg* 73, 6, juin 1947
- Gauthier Villars (P) et Dontzoff (A) Anatomie macroscopique du cancer de l'œsophage *Presse méd* 20 sept 1947
- Didier (G) Les apports de l'endoscopie en chirurgie cancéro œsophagienne *J des Sc méd de Lille* 65 7 sept 1947
- Huguenin Brulé et Rose Cancres trachéo et broncho œsophagiens *Soc franç de pathol resp* 12 oct 1947
- Rebattu et Martin Cancer de l'œsophage ouvert dans l'aorte *Soc d O R L de Lyon* III de la Région séance du 17 déc 1947
- Mayoux et Meyer Métastase cutanée dans un épithélioma de l'œsophage *Soc d O R L de Lyon et de la Région*, séance du 17 déc 1947
- Minet Linquette Desruelles et Goudemand Cancer de l'œsophage communiquant largement avec un abcès pulmonaire *Soc de méd du Nord* 19 déc 1947
- Baer (P) et Siche (K) L'association d'achalasie du cardia et du cancer de l'œsophage *Brit J Radiol* 2, 240 déc 1947, pp 489-540
- Taquino (G J) et Joseph (G F) Cancer de l'œsophage Etude de 145 cas avec étude partielle des métastases et de l'extension *Ann Otol, Rhinol and Laryngol* 56 4 déc 1947, pp 1041-1058
- Resano (J H) Réflexions sur les difficultés de diagnostic des lésions œsophagiennes *Anal de oto rino laryngol del Uruguay* Montevideo, 16 4 1947 pp 287-305
- Ogilvie (H) Le diagnostic précoce du cancer de l'œsophage et de l'estomac *Brit Med J* no 4523 1947 pp 405-407
- Buttkofer (R) L'irradiation rotatoire du cancer de l'œsophage *Radiol clin*, Bale 16 1 1947 pp 7-12
- McPeak (E) et Arons (L Walter) Adenoacanthoma of the esophagus *Arch Path* 44 1947 pp 385-390
- Adams (H) Tumeurs primitives de l'œsophage *Surg Clin North Amer* 27 3 1947
- Pearlman (S J) Le prétendu carcino sarcome de l'œsophage *Ann Oto-Laryngo* 64 7, 1947 p 372
- Doyle (L) Carcinome de l'œsophage et de la portion cardiaque de l'estomac *Austral and New Zeal J Surg* 17 1 1947, pp 16-28
- Tanew (N) Contribution au diagnostic des tumeurs polypoides de l'œsophage *Wien klin Wochenschr* 59 45 1947, pp 745-750
- Hudson (W A) Carcinome de l'œsophage Quelques considérations sur deux observations *Ann Oto Laryngol*, 64 7 1947 pp 385-386
- Szenes Idiopathische Ösophagusdilatation und Karzinom *Monatsschr f Ohrenh und Lar* 81 2 1947 pp 100-102
- Benedict (E B) A propos du traitement du cancer de l'œsophage par la chirurgie la radiothérapie et le bougrage *Ann Oto Laryngol* 64 7 1947 p 374
- Auguste (C) et Paris (J) Cancer développé sur un méga œsophage évoluant depuis 24 ans *Gaz méd de France* 53 5 mars 1947 in *J de chir* 63 3 1947
- Léger A propos du traitement du cancer de l'œsophage *Acad de chir*, 18 déc in *J de chir* 63 7-8 1947
- Rebattu Deux cas de syndrome de Cl Bernard Horner au cours du cancer de l'œsophage *Lyon méd* 78 48 1 déc in *J de chir* 63 6 1947
- Schlörhauser (W) et Mellauner (A) Cancer du diverticule de Zenker *Der Krebskr* Vienne 2 4 1947 pp 155-164
- Desgrez et Antoine Renseignements fournis par l'examen radiologique dans la cure chirurgicale des cancers de l'œsophage *Soc franç d electroradiol nat*, 13 janv 1948

- Muller (B) et Michel (J) Un cas de cancer du cardia développé au cours d'un méga-œsophage
Lyon m'd 75 4 25 janv 1948
- Jousseume Les formes respiratoires du cancer de l'œsophage *Ouest m'd* Paris 1 1 janv 1948 pp 1-24
- Lefèvre Le traitement radiothérapique du cancer de l'œsophage *Paris méd* 38 11 20 mars 1948 pp 137-152
- De Bakey (M. E.) et Ochsner (A.) Carcinoma of the esophagus *Post Graduate Med* 3 mars 1948 pp 192-198
- Freireich (A.) Cancer de l'œsophage sans dysphagie *New York State J Med* 48 8 15 avril 1948
- Dor (J.) L'orientation moderne du traitement du cancer de l'œsophage *Sud méd et chir* 80 30 avril 1948
- Portmann (G.) et Lantillac (J. P.) Cancer de l'œsophage étendu envahissant la muqueuse en surface *Soc d'orth et de chir de l'app mot de Bordeaux in France méd* no 4 avril 1948
- Carrillo Maurtua Cancer de l'œsophage *Inter-Exter* Maloigne no 12 avril 1948
- Nemours Auguste A propos du cancer de l'œsophage *Soc nat franç de gastro-entérol* 10 mai 1948 in *Arch d mal de l'app dig* 37 1948
- Chene (P.) et Menoua Remarques pour le diagnostic précoce du cancer de l'œsophage *Presse méd* 15 mai 1948
- Buch (A.) Technique du traitement du cancer de l'œsophage par tubes de radium mis en place sous contrôle œsophagoscopique *Revue de L. O. R.* Bordeaux no 5-6 mai juin 1948
- Banche (M.) Syndrome de Pancoast Tobias par cancer primitif de l'œsophage *Minerva méd* Turin 39 25 23 juin 1948
- Watson (W. L.) et Pool (J. L.) Cancer de l'œsophage cervical *Surgery* 23 6 juin 1948
- Sweet (R. H.) Le carcinome de l'œsophage et de l'estomac *J A M A* 137 15 31 juin 1948 pp 1213-1219
- Raven (R.) Carcinome de l'œsophage étude clinique et pathologique *Brit J Surg* Bristol 36 141 juil 1948
- Hillemand (P.) Le Brigand et Coffin A propos du diagnostic précoce du cancer de l'œsophage *Arch des mal de l'app dig* 37 7-8 juil août 1948 pp 516-518
- Levrat (M.) et Despierrres (G.) A propos des fistules œsophago-aériennes dans le cancer de l'œsophage *J de méd de Lyon* no 688 5 sept 1948
- McPeak (E.) et Warren (S.) Aspects histologiques des cancers de l'œsophage juxta-cardiaque et des cancers du cardia *Am J Pathol* 24 5 sept 1948 pp 971-1001
- Nemours Auguste Cancer de l'œsophage *Arch des mal de l'app dig* 37 9-10 sept-oct 1948 pp 601-606
- Resano (J. H.) et Albanese (A.) (Buenos Aires) Sarcome primitif de l'œsophage *Ret. de chir* 67 9-10 sept-oct 1948 pp 257-274
- Hétroy (M.) Essais de traitement des cancers du larynx et de l'œsophage chez l'homme par les estropènes de synthèse *Concours méd* 13 nov 1948
- Sweet (R. H.) Le diagnostic et la conduite générale du traitement du carcinome de l'œsophage *Rocky Mount Med J* 45 1 nov 1948 pp 931-934
- Lindert (M. C. F.) et Winnik (D.) Perforation of the esophagus a complication of carcinoma of the esophagus *Am J Dig Dis* 15 11 nov 1948 pp 378-381
- Guéret (A.) et Lambling L'exploration tomographique de la région cardio-tubé rositaire et de l'œsophage abdominal Son application dans le diagnostic du cancer *Soc nat franç de gastro Entérol* 13 déc 1948 in *Presse méd* no 12 19 fév 1949
- Ziegler (H.) La péritonite par propagation dans le cancer de l'œsophage voisin du cardia *Der Krebsarzt* Vienne 3 12 déc 1948
- Fingelstad (R. B.) Carcinoma œsophagi helbredet ved stralebehandling *Tskr Norske Lægefor* 68 1948 p 272
- Stephens (H. H.) Cancer de l'œsophage *California Med* 68 5 1948 pp 388-391
- Desgrez et Antoine Examen radiologique dans la cure chirurgicale des cancers de l'œsophage *J radiol electr* Paris 29 5-6 1948
- Mc Gregor (Lee) Ulcer and cancer of esophagus stomach duodenum and pancreas *S Afric Med J* 22 1948 pp 214-215
- Borak (J.) Strahlentherapie des Oesophaguscarcinoma *Strahlenther* 77 1948 pp 349-354
- Grez Cancer de l'œsophage *Rev. de oto-rhino laringol* Santiago 8 4 1948 pp 265-276
- Pack (G. T.) *Cancer of the esophagus and gastric cardia* C. V. Mosby Company St Louis 1949
- Auguste (C.) et Heraud (M.) Cancer inoperable de l'œsophage évoluant depuis plus d'un an

- sous une forme douloureuse et non sténosante *Soc de gastro-entérologie de la Région du Nord*, 6 fév 1949 in *Presse méd*, 78 60 24 sept 1949
- Eerland Le diagnostic précoce du cancer de l'œsophage *Nederl tijdschr voor geneesk*, 93 13 26 mars 1949 pp 923-930
- Mogena (H) Cancer de l'œsophage *Prensa méd argent*, 36, 13, 1 avril 1949
- Purdy Stout (A), Humphreys (G) et Rottenberg (L. A.) Un cas de carcino-sarcome de l'œsophage *Am J Roent and Radiumther*, 61, 4 avril 1949 pp 461-469
- Hawthorne (H. R.) et Froese (A. S.) Le cancer de l'œsophage thoracique *Rec Gastro Enterol* 16 4, avril 1949
- Anderson McDonald et Olsen Diagnostic cytologique de cancer de l'œsophage et du cardia *Proc Staff Meet, Mayo Clin*, 11 mai 1949
- Dutif et Limousin Linite plastique avec métastase œsophagienne *Soc des Sc méd de Clermont Ferrand*, 12 mai 1949
- Lutz (P) Radiographie inhabituelle de l'œsophage dans le cancer du fundus gastrique *Der Krebsarzt* 4 5-6 juin 1949
- Dani (W) L'œsophago-gastrostomie dans le cancer inopérable du cardia *Der Krebsarzt*, Vienne 4, 5-6 juin 1949
- Brick (I) Hernie du hiatus et cancer de l'estomac et de l'œsophage *Gastro Enterol* 15 7 juil 1949
- Antoine (M) et Massol (M) Radiologie du cancer de l'œsophage opéré *Sem d hôp de Paris* 25 54 22 juil 1949
- Crump (A. B.) et Nennig (G. C.) Le diagnostic du cancer de l'œsophage La méthode de biopsie avec la curette rétrograde *Gastro Enter*, 13 2 août 1949 pp 153-160
- Zinninger (M. M.) et Collins (W. T.) L'extension du cancer de l'estomac au duodénum et à l'œsophage *Ann Surg*, 130 3 sept 1949
- Cottet (P) Tumeurs exceptionnelles de l'œsophage sarcome polypeux et fibrome sténosant *Radiol clin. Bale*, 18 5 sept 1949
- Courty (L.) et Langeron (P) Sur un cas de cancer sus-cardial et juxta-œsophagien de la grosse tubérosité gastrique avec rétrécissement de la portion terminale de l'œsophage *Arch des mal de l'app dig Paris* 38 9-10 sept-oct 1949
- Mouchet (A.) Un cas de néoplasme œsophagien développé sur un brachy-œsophage *Ann de chir*, 19 oct 1949
- Terracol (J.) et Guertier (Y.) Le diagnostic du cancer de la bouche de l'œsophage *XXIII Congrès franç d'oto-rhino-laryngol* 20 oct 1949
- Terracol (J.) Le diagnostic précoce du cancer de l'œsophage *Concours méd* 29 oct 1949
- Krebs (C.) Nielsen (H.) et Andersen (P.) Le traitement radiothérapique tournant du cancer de l'œsophage observations cliniques *Acta radiol*, Stockholm 32 4, 31 oct 1949
- Hicquet (M.) Diagnostic du cancer du cardia *Ann d'oto-laryngol*, 66 10-11, oct nov 1949
- Imbrigha (J. E.) et Iopusnaki (M. S.) Cytologic examination of sediment from the esophagus in a case of intra epidermal carcinoma of the esophagus *Gastro Enterol* 13 5 nov 1949 pp 457-463
- Lehman (D. J. Jr) et Hawkes (S. Z.) La maladie de Hodgkin de l'œsophage *Gastro-Enterol*, Baltimore 13 5 nov 1949
- Lortat Jacob (J. L.) et Badaro Deux sarcomes primitifs de l'œsophage pédiculés *Résumés chir de l'hôp Broussais* 12 déc 1949 in *Sem des hôp*, 18 mai 1950
- Chene (P.) et Merroua (A.) Une observation du cancer de l'œsophage *Gaz méd de France* 57 24, 25 déc 1949
- Chene (P.) Fauvet (J.) et Chavy (J.) La clinique du cancer de l'œsophage *Gaz méd de France* 57 24 25 déc 1949
- Chêne (P.) et Markovits (P.) Les difficultés radiologiques dans le diagnostic précoce du cancer de l'œsophage thoracique *Gaz méd de France* 57 24 25 déc 1949
- Brulé (G.) Les endoscopies dans le cancer de l'œsophage *Gaz méd de France* 57 24 25 déc 1949
- Orcel (L.) Etude de l'extension œsophagienne du cancer gastrique *Thèse Paris* 1949
- De Reynier (J. P.) A propos d'un cas de fistule cancéreuse œsophago-trachéale *Practica oto-rhino-laryngol* 11 5-6 1949
- Gros (C. M.) Fruhling et Speeg Du cancer de l'œsophage *J de radiol d'électrol et Arch d'électr méd Paris* 30 11-12 1949
- Leroux (L.) Les tumeurs de l'œsophage *Bronchosc œsophagosc et gastrosc* an 1949
- Mallory (T. B.) Castleman (B.) et Tome (W. W.) Fibrosarcome de la glande thyroïde Canc. pavimenteux de l'œsophage *Acta Erlang J Med* 74 5 2 fév 1950
- Scalone (I.) Syndrome gastrique pré-dysphagique dans le cancer de l'œsophage *Presse méd*, 58 12 25 fév 1950

- MacDougall Cancer *J de chir* 66 2 fév 1950
- Byrnes (C K.) Le cancer de l'œsophage *Irish J Med Sc* Dublin 6 290 fév 1950
- Cottet (P.) Tumeur exceptionnelle de l'œsophage sarcome polypeux et fibrome sténosant
Radiol gica clin Bâle 18 sept 1949 pp 258-265
- Pescetti (V.) Paralisi laringea destra precoce in un cancro esofages asintomatico *Il Valsala*
26 1 fév 1950
- Puyo (R.) et Portmann (M.) Mélano-carcinome primitif de l'œsophage *Rev de laryngol*
Bordeaux mars avril 1950
- Robaud (P.) Etude de l'action des œstrogènes de synthèse sur les cancers du larynx et de
l'œsophage *Thèse Paris* 1950
- Clark (D E.) Sarcoma of the esophagus Report of successful resection of a fibrosarcoma
Arch Surg Chicago 59(2)348-354 1949
- Lortat Jacob (J L.) and Badaro Deux sarcomes primitifs de l'œsophage pédiculés (Two
pedunculated primary sarcomas of the esophagus) *Sem hop* Paris 26(37) 1743-1746
1950
- Thorek (P.) and Neuman (B H.) Rhabdomyosarcoma of the esophagus *J Thoracic Surg*
20(1) 77-89 1950
- Lions (A S.) and Garlock (J H.) Leiomyosarcoma of the esophagus Report of first success-
ful resection *Surgery* 29(2) 281-287 1951
- Mauro (E.) and De Escobar (C T.) Sarcoma do esofago (Sarcoma of the esophagus) *Arch*
Cirurg clin exp 14(2) 69-80 1951
- Chalmagne (J.) Un cas de cancer mélanique de l'œsophage (A case of melanocarcinoma of the
esophagus) *Acta oto rhino laring belg* 5(1) 76-79 1951
- Owens (J M.) and Russell (W O.) Concurrent leiomyosarcoma and squamous carcinoma of
the esophagus *Arch Path* 51(5) 560-564 1951
- Morson (B C.) and Belcher (J R.) Adenocarcinoma of the esophagus and ectopic gastric
mucosa *Brit J Cancer* 6(2) 127-130 1952
- Resano (J H.) and Hojman (D.) Contribución al estudio del sarcoma primitivo di esofago
(Contribution to the study of primary esophageal sarcoma) *Prensa méd argent* 39(35)
2045-2056 1952
- Messelt (O T.) Cytological diagnosis of esophagus cancer *Acta chir scand* 103(6) 440-441
1952
- Neuberger (F.) Über die lymphogenen Schleimhautmetastasen der Speiseröhrenkreise
(Lymphogenous mucosal metastases of esophageal cancer) *Wien klin Wschr* 65(39)
821-823 1953
- Thompson (J R.) Carcinosarcoma of the esophagus Report of a case with autopsy findings
J Thoracic Surg 25(3) 261-264 1953
- Badosa Caspar (J.) and Boado (O L F.) Leiomyosarcoma del esofago (Leiomyosarcoma of the
esophagus) *Rev esp Enferm Apar dig* 12(2) 173-185 1953
- Mainiero (D.) Stenosi esofagea da compressione di metastasi linfoghiandolari mediastiniche
per tumore rarissimo dell'esofago (raddomyosarcoma) (Structure of the esophagus from
compression by mediastinal lymph node metastases of an extremely rare tumor of the
esophagus (rhabdomyosarcoma)) *G ital chir* 10(4) 301-315 1954
- Boyd (D P.) Meissner (W A.) Velkoff (C L.) and Gladding (T C.) Primary melano-
carcinoma of the esophagus Report of a case *Cancer* 7(2) 266-270 1954
- Wurmig (P.) Über krebsdisponierende Zustände an Ösophagus und Kardia (Conditions in
esophagus and cardia predisposing to cancer) *Arch klin Chir* 780(5) 504-515 1955
- Loring (W E.) and Zeppa (R.) Melanocarcinoma of the esophagus *J Thoracic Surg*
32(1) 35-45 1956

Chapter 28 : Surgical Treatment of Carcinoma

- Ballivet (M.) La chirurgie radicale du cancer de l'œsophage thoracique *J de chir* 55 2
fév 1940
- Garlock (J H.) Surgical treatment of carcinoma of the thoracic esophagus *Surg Gynec et*
Obst 70 2 A fév 1940 pp 556-569
- Carter (B N.) Stevenson (J.) et Abbott (O A.) Transpleural esophagogastronomy for
carcinoma of the esophagus and for carcinoma of the cardiac portion of the stomach
Surgery 8 oct 1940 pp 587-603
- Carter (B N.) Resection of the portion of thoracic esophagus for carcinoma report of two
cases *Surg Gynec and Obst* 71 nov 1940 pp 624-632
- Garlock (J H.) Surgical treatment of carcinoma of the esophagus *Arch Surg* 41 nov 1940
pp 1184-1214

BIBLIOGRAPHY

660

- Adams (W E) Pathologic considerations relating to early diagnosis and curative surgical treatment of carcinoma of the esophagus *Surg Gynec and Obst*, 77 fév 1941 pp 105-110
- Adams (W E) Recent progress in surgical treatment of carcinoma of the esophagus *Revue of Gynec and Obst* 72 fév 1941 pp 312-317
- Santy (P) et Balliv (M) L'oesophagectomie thoracique pour cancer Technique et résultats actuels *Presse méd* 19-22 fév 1941
- Ochsner (A) et De Bakey (M) Surgical aspects of carcinoma of the esophagus *Review of literature and report of 4 cases J Thoracic Surg* 10 avril 1941 pp 401-445
- Phemister (D B) Operative treatment of carcinoma of the thoracic esophagus *Illinois Med J* 79 juin 1941 pp 497-500
- Garlock (J H) Present status of the surgical treatment of carcinoma of the thoracic esophagus case report with description of a new artificial esophagus *Ann Otol Rhinol and Laryngol* 50 déc 1941 pp 1228-1232
- Santy (P) Balliv (M) et Bérard (M) Préparation opératoire et traitement palliatif des cancers digestifs par le Fernescorbone sodique *J méd de Lyon* 1941 p 526
- Ivanusovich (O) et Ferran (R C) Carcinoma of the esophagus Tork operation Recovery *Surg Gynec and Obst* 74, janv 1942 pp 47-52
- Jonas (A F Jr) Transpleural esophagostomy report of a successful case *Arch Surg* 44, mars 1942 pp 556-561
- Walters (W) Transthoracic resection of stomach and esophagus, case *Proc Staff Meet Mayo Clin* 17 22 avril 1942 pp 241-247
- Phemister (D B) Experiences with 8 cases of resection of the esophagus review of 21 *Thoracic Surg*, 11 juin 1942, pp 484-493
- Stephens (H) Cancer of the thoracic esophagus and upper end of the stomach review of 21 cases that have undergone surgical exploration *J Thoracic Surg*, 11, juin 1942 pp 469-483
- Wu (Y K) et Loucks (H H) Resection of the esophagus for carcinoma Analysis of experiences in 11 cases *J Thoracic Surg* 11 juin 1942 pp 516-528
- Churchill (L D) et Sweet (R H) Transthoracic resection of tumors of the stomach and esophagus *Ann Surg*, 115 juin 1942 pp 897-920
- Holman (C W) et Mc Swain (B) Transthoracic esophago gastrectomy *Surgery* 11 juin 1942 pp 882-885
- Meyer (H W) Intrathoracic esophagostomy for total gastrectomy with lower esophagectomy for carcinoma *Surgery* 12 juin 1942 pp 115-127
- Franklin (R H) 2 cas d'ablation heureuse de l'oesophage thoracique pour cancer *Brit J Surg*, Londres 30 117, juin 1942
- Hermann L Emphyseme médiastinale avec le pneumothorax sous tension bilatérale comme complication des strumectomies et des interventions pour cancer de l'oesophage *Zentralbl fur Chir* 68 44 31 oct 1942
- Allison (P R) 4 cas de carcinome de l'oesophage traités par l'excision *Brit J Surg* Londres no 118 oct 1942
- Laumonier (M P) Présentation d'un malade guéri opératoirement depuis six mois d'une tumeur de l'oesophage thoracique *Soc de chir de Bordeaux* 22 nov 1942
- Sweet (R H) Surgical management of carcinoma of the mid thoracic esophagus *Tr Am Acad Ophth and Otolaryng* 47 nov dec 1942 pp 75-91
- Kay (C B) Experimental observations on reconstructive intrathoracic esophagostomy anastomosis following resection *Surg Gynec and Obst* 76 mars 1943 pp 300-314
- Adams (H D) Rational approach to resection of carcinoma of the upper two thirds of the thoracic esophagus *Lakey Clin Bull* 3 avril 1943 pp 121-123
- Pack (G T) et Watson (W L) Transthoracic subtotal gastrectomy and esophagectomy *Arch Surg* 46 juin 1943 pp 930-938
- Santy (P M) Balliv (M) et Bérard (M) Transthoracic esophagectomy for cancer report of 2 successful cases *J Thoracic Surg* 17 juin 1943 pp 397-431
- Swenson (O) et Magruder (Th) Jr Esophagectomie expérimentale *Surgery*, 18 22 sept 1943 pp 337-344
- Miscall (L) et Clark (B) Le bouton de Murphy dans l'oesophagostomie *Surgery*, 14 6 juin 1943
- Clagett (O T) et Wang (L M) Transthoracic resection of esophagus and stomach for carcinoma 2 cases *Proc Staff Meet Mayo Clin* 18 22 sept 1943 pp 337-344

- Brea (M M) Surgical therapy of cancer of thoracic esophagus *Medicina Buenos Aires* 4 1943 p 74
- Garlock (J H) Reestablishment of esophago gastric continuity following resection of the esophagus for carcinoma of the middle third *Surg Gynec and Obst* 78 janv 1944 pp 23-28
- Dorsey (J M) Malignant tumors of the cardiac end of the stomach and terminal esophagus *Surg Clin North Amer* 24 fév 1944 pp 225-237
- Adams (R) Carcinoma of the thoracic esophagus clinical observations and experiences with 28 surgically explored cases *Surg Clin North Amer* 24 juin 1944 pp 684-693
- Santy (I) Ballivet (M) et Latarjet (M) Technique et résultats de l'œsophagectomie thoracique pour cancer Voie transpleurale droite avec œsophagoplastie secondaire par transposition préthoracique de l'estomac *Lyon chir* 39 5 sept-oct 1944 pp 121-122
- Garlock (J H) Causes of mortality following radical resection of the esophagus for carcinoma *J Thoracic Surg* 13 oct 1944 pp 415-423
- Lortat Jacob et Mathey (J) Rétablissement immédiat de la continuité œsophagienne après œsophagectomie segmentaire pour cancer *Acad de Chir* 18 oct 1944 pp 1 2 3 4
- Orsoni (P) Le rétablissement de la continuité du tube digestif dans les œsophagectomies pour cancer œsophagoplastie abdomino-médiastino-cervicale par tube gastrique *Acad de chir* 22 nov 1944
- Vasconcelos (E) Original technic for total esophagectomy *Dis of Chest* 10 nov-déc 1944 pp 471-480
- Garlock (J H) Diagnosis and surgical treatment of cancer of the esophagus *Proc Intern Postgrad M A North Amer* (1943) 1944 pp 186-190
- Thomsson (V) Carcinome de l'œsophage résection et œsophagogastronomie *Brit J Surg* 37 127 janv 1945
- Taylor (H) Une opération pour l'ablation du carcinome de l'œsophage gastrostomie pré sternale *Brit J Surg* 32 127 janv 1945
- Clark (D E) Transthoracic esophagogastronomy for carcinoma of the middle third of the esophagus successful resection *Ann Surg* 171 janv 1945 pp 65-73
- Sweet (R. H) Transthoracic gastrectomy and esophagectomy for carcinoma of the stomach and esophagus *Clinics* 3 fév 1945 pp 1288-1315
- Sweet (R. H) Transthoracic resection of the esophagus and stomach for carcinoma Analysis of postoperative complications causes of death and late results of operation *Ann Surg* 121 mars 1945 pp 272-284
- Bradshaw (H H) et O'Neill (J F) Surgical treatment of some lesions of the lower esophagus and upper stomach *J Thoracic Surg* 14 juin 1945 pp 187-194
- Brantigan (O C) Anastomosis following resection of the middle third of the esophagus for carcinoma *Bull School of Med Univ Maryland* 30 juil 1945 pp 32-38
- Sweet (R H) Surgical management of carcinoma of the midthoracic esophagus preliminary report *New England J Med* 273 5 juil 1945 pp 1-7
- Lewis (I) Deux cas de cancer de l'œsophage traités par la résection avec œsophagogastronomie par voie transpleurale *Proc Roy Soc of Med* no 9 juil 1945 pp 482-483
- Kross (I) Carcinoma of the esophagus Transpleural resection and esophagogastronomy *Am J Digest Dis* 12 oct 1945 pp 344-346
- Santy et Ballivet Œsophagectomie thoracique pour néoplasme Anastomose œsophagogastrique intrathoracique *Soc de chir de Lyon* 6 déc 1945
- Steele Carcinome de l'œsophage *Lancet* no 2 111 dec p 121 *Int J de chir* 61 10-12 1945
- Maria de Freitas (J) Résection transthoracique de l'œsophage et de l'estomac *Ann du IF Coneres med de Sao-Paulo Brésil* 1945 p 794
- Santy Ballivet et Maillet Cancer du tiers moyen de l'œsophage œsophagectomie transpleurale droite avec rétablissement immédiat de la continuité par transposition thoracique de l'estomac *Acad de chir* 16 janv 1946
- Lainé (E) Cancer de l'œsophage thoracique opéré avec succès *Soc de gastro-entérol de Paris* (région du Nord) 20 janv 1946 in *Arch d mal de l'app dig* 35 1946
- Thompson (V C) Œsophagectomie pour cancer Transplantation antéthoracique jéjunale *Proc Roy Soc of Med* 39 11 avril 1946
- Clagett (O T) Transthoracic resection of lesions of lower part of esophagus and cardia of the stomach *Texas State J Med* 47 7-11 mai 1946
- Ingebrigsten (R) Carcinome de l'œsophage partie thoracique excision guérison opératoire *Acta chir Scand Stockholm* 94 1-2 15 juin 1946
- Lewis (Ivor) Le traitement chirurgical du cancer de l'œsophage avec mention spéciale d'une nouvelle opération pour les tumeurs du tiers moyen *Brit J Surg* 34 133 juil 1946 pp 18-31

- Lawrence (E A) Transthoracic resection of the esophagus for carcinoma *Rocky Mountain Med J* 43, juil 1946, pp 547-553
- Humphreys (G H) Etude des résections du cancer œsophagien et gastrique *Ann Surg* 124, 2 août 1946, pp 288-299
- Balliv et (M) Œsophago gastrectomie par voie thoracique gauche pour cancer haut situé de la petite courbure, propagé au cardia *Acad de chir* 16 oct 1946 (rapport d'Affaires et discussion)
- Sweet (R H) Cancer de l'œsophage thoracique moyen. *Ann Surg*, 124, 4 oct 1946 pp 653-666
- Carter (B N) et McGrath (F J) Esophagogastrostomy for lesions of the upper stomach and lower end of the stomach *Surg Clin North Amer* 26 oct 1946 pp 1125-1139
- De Vernejoul Métras et Ballivet Œsophagectomie pour cancer de l'œsophage thoracique *Acad de chir* 72 27 nov 1946 p 513
- Nagel (G W) et Menke (J J) Transthoracic operations for neoplasms of the esophagus and stomach *Surg Gynec and Obst* 83, 3, nov 1946 pp 657-666
- Winkelbauer (Gratz) Sur la technique de la plastie cutanée après résection du cancer de l'œsophage cervical *Der Krebsarzt*, 1 11 nov 1946 pp 475-482
- Wells (H) Traitement chirurgical du cancer de l'œsophage *Acad de chir* 4 déc 1946
- Garlock (J H) La voie d'abord abdomino thoracique pour la résection des cancers du cardia et de la partie basse de l'œsophage *Surg, Gynec and Obst* 83, 6 déc 1946, pp 737-741
- Mautz (F R) Transpleural resection of the esophagus and stomach for esophageal obstruction *Ohio State M J* 47 déc 1946 pp 1243-1247
- Rienhoff (W F) Jr Intrathoracic esophagojejunostomy for lesions of the upper third of the esophagus *South m Med J* 39 déc 1946 pp 928-940
- Allison (P R) Œsophago jejunostomie pour cancer inopérable du cardia *Thorax*, 1, 4 1946 pp 239-246
- Lortat Jacob (J L) et Mathey Cancer de l'œsophage thoracique succès opératoire *Ann Oto Laring* 13 9-10 1946 pp 453-457
- De Vernejoul et Métras (H) Œsophagectomies pour cancers de l'œsophage thoracique anastomoses gastro œsophagiennes trois observations *Mém Acad de chir de Paris* 72 29-30-31 1946 pp 513-532
- Christoffersen (J C) Sur le traitement chirurgical du cancer de l'œsophage et du cardia *Nord med* 33 3 10 janv 1947 pp 185-189
- Mouchet (A) Cancer de l'œsophage haut situé à 1 cm 1/2 sous la clavicule *Acad de chir* 14 janv 1947
- Lortat Jacob Œsophagectomie pour cancer avec rétablissement immédiat de la continuité digestive *Acad de chir* 14 janv 1947
- Hertzog (P) et Coenilhez (A) Etat actuel de la chirurgie d'extérèse du cancer de l'œsophage *Rev de chir* 66 3-4 mars avril 1947
- Brunschwig (A) Résection en un temps de la totalité de l'œsophage cervical *Laryngoscope* 57 mai 1947, pp 305-312
- Garlock (J H) Considérations techniques sur le traitement chirurgical du cancer de l'œsophage et de la partie haute de l'estomac *J Thoracic Surg* 16 3, juin 1947 pp 215-253
- Brown (C O) Le traitement chirurgical du cancer de l'œsophage *Med J Austral Sydney*, 74 2 1 5 juil 1947
- Nicole (R) L'opération du carcinome de l'œsophage *Schweizer med Wochenschr*, Bâle 77 27-28 12 juil 1947
- Rouget (J) Chirurgie du cancer de l'œsophage *Ann d'O R L* oct 1947 p 575
- Ingebrigsten (R) Cancer du cardia et de l'œsophage résection intrathoracique gastrique et œsophagienne, œsophago gastrostomie *Nordisk med Stockholm* 35 47 21 nov 1947
- Santy ■ Mouchet Traitement chirurgical du cancer de l'œsophage thoracique *Congrès fran, de chir* Paris oct 1947
- Mouchet (A) et Rouget (J) A propos de la chirurgie du cancer de l'œsophage thoracique *Soc d'O R L séance du 15 déc 1947*
- Rudler Cancer du cardio-œsophage enlevé par voie thoracique gauche guérison opératoire *Acad de chir* 17 déc 1947
- Dor (J) et Claustre (P) Cancer de l'œsophage thoracique moyen avec extérèse suivie du rétablissement immédiat de la continuité *Soc de chir de Marseille* 22 déc 1947
- Woodward (F D) Le traitement chirurgical du cancer œsophagien cervical et rétro-cœcien *Ann Otol Rhinol and Laryngol* 56 4 déc 1947, pp 1035-1040
- Maria de Freitas (J) Fanganelo (M) et De Mello (F F) Le cancer de l'œsophage sur 14 cas opérés *Anais Paulista de med e cir* Sao-Paulo 54 6 déc 1947, pp 391-405

- Sweet (R. H.) Progrès dans le traitement chirurgical du cancer de l'œsophage *Surgery* 84 1947 pp 373-374
- Courbez (L.) Le traitement chirurgical du cancer de l'œsophage *J d Sc méd de Lille* 65, 7 sept 1947
- Ugon (V. A.) Leguisamo (A.) et Ugon (R. A.) L'exstirpation du cancer de l'œsophage thoracique *Arch urug de Med, Cir y Especialid* Montevideo 16 4 avril In *J de chir*, 63, 9 1947
- Eerland (L. D.) Quelques observations sur l'œsophagectomie transthoracique en cas de cancer de l'œsophage *Bruxelles méd* 77 34 1947 pp 1839-1847
- Brunschwig (A.) et Camp (E.) Résection en un temps de la totalité de l'œsophage cervical du larynx de la base de la langue de la trachée cervicale et d'une double chaîne ganglionnaire pour cancer primitif de l'œsophage cervical. Reconstitution de l'œsophage cervical *Laryngoscope* 57 5 1947 pp 305-312
- Taylor (H.) Cancer de l'œsophage traité par résection et œsophago-gastrostomie pré sternale *Proc Roy Soc Med* 40 8 1947 pp 465-466
- Pinkham (R. D.) Résection transthoracique du cancer de la moitié supérieure de l'œsophage *West J Surg* 55 3, 1947 pp 147-151
- Courty (L.) Le traitement chirurgical du cancer de l'œsophage. L'œsophagectomie intra thoracique *J Sc méd de Lille* 65 7 1947 pp 123-130
- Segers (A. M.) Cancer du tiers moyen de l'œsophage. Considérations sur son traitement *Prensa med argent* 34 46 1947 pp 2182-2185
- Wilton (J.) Total esophagectomy for carcinoma of the middle third of the esophagus. Report of a successful operation *S Africa Med J* 21 1947 pp 944-946
- Garlock (J. H.) Progress in the surgical treatment of carcinoma of the esophagus and upper stomach *Ann Roy Coll Surg* 2 1947 pp 183-188
- Sweet (R. H.) Cancer de l'œsophage et de l'extrémité cardiaque de l'estomac. Résultats immédiats et tardifs du traitement par résection avec anastomose œsophago-gastrique *J A M A* 135, 8 1947 pp 845-890
- Johnson (J.) Traitement chirurgical du cancer de l'œsophage *Ann Oto Laryngol* 64 7 1947 p 374
- Sapozhkov (K.) L'exclusion de l'œsophage dans les cancers et les sténoses cicatricielles de sa portion thoracique *Chirurgica* Moscou no 4 1947 In *J de chir* 63 12 1947
- Huun (Yvan) Contribution à l'étude du traitement chirurgical du cancer du cardio-œsophage *Thèse Nancy* 1947
- Léger (I.) et Tricard (A.) Exérèse d'un cancer haut situé de l'œsophage thoracique avec rétablissement de la continuité *Bulletin méd* no 2 11 janv 1948
- Olivier (Cl.) Place de la tunnellisation cervico-abdominale dans le traitement du cancer de l'œsophage *Presse méd* 56 1 janv 1948
- Wookey (Harold) Le traitement chirurgical des cancers de l'hypo-pharynx et de l'œsophage *Brit J Surg* 35 139 janv 1947 pp 249-266
- Hertzog Suites simples d'une intervention pour cancer de l'œsophage se situant au niveau de la bifurcation traité par résection avec anastomose gastro-œsophagienne *B A C* janv 1948
- D Allaines (I.) et Dubost (Ch.) Sur la chirurgie de l'œsophage thoracique *Acad de chir* séance du 10 fév 1948
- Mouchet (A.) et Orsoni A propos de 7 résections de l'œsophage pour cancer avec anastomose œsophago-gastrique suivie de guérison opératoire *Acad de chir* 18 fév 1948
- Rudler A propos de la chirurgie du cancer de l'œsophage *Acad de chir* séance du 25 fév 1948
- Lortat-Jacob Quelques interventions pour cancer de l'œsophage *Soc nat franç de gastro-entérologie* 8 mars 1948
- Rudler (J. C.) Les possibilités actuelles de la chirurgie du cancer de l'œsophage thoracique et du cardia *Soc de méd de Paris* 26 mars 1948
- D Allaines (G.) et Dubost (A.) Trois ans de chirurgie de l'œsophage thoracique *Soc nat franç de gastro-entérologie* 12 avril 1948
- De Costa (J. F.) (Lisbonne) Traitement chirurgical du cancer de l'œsophage thoracique par résection suivie d'œsophago-gastrostomie *J de med* Lisbonne 11 273 17 avril 1948 p 401 et 274 24 avril 1948 pp 429-435
- Carlock (G. H.) Les progrès dans le traitement chirurgical du cancer de l'œsophage et de la partie supérieure de l'estomac *Ann Roy Coll Surgeons of England* 2 2 avril 1948 pp 165-218
- Strieder (J. W.) Traitement chirurgical du cancer des 2/3 supérieurs de l'œsophage et du cardia *J Thoracic Surg* 17 2 avril 1948 pp 143-161

- I éger (L.) Adaptation de la voie de Fiolle et J Delmas à l'abord des cancers de l'œsophage cervico thoracique *Technique de Resano Presse méd* 9 mai 1948, pp 329-335
- Mouchet (A.) Traitement chirurgical du cancer de l'œsophage thoracique *Concours méd* 8 mai 1948 in *Arch d mal de l'app dig* 37 1948
- Velasco (R. N.) Cancer du tiers moyen de l'œsophage. Résection et anastomose œsophago-gastrique supra aortique *Rev de la Assoc med argent*, no 629-630 15-30 mai 1948 pp 256-258
- Soler Raig (Barcelone) et Chalmot La chirurgie transthoracique de l'appareil digestif *J chir internat de Turin*, 27, 28 et 29 mai 1948
- Dor (M. J.) Deux pièces d'œsophagectomie, l'une pour cancer l'autre pour rétrécissement du 1/3 inférieur de l'œsophage *Guerison Soc de chir de Marseille séance du 14 juin 1948*
- Govaerts (J.) et Coryn (J.) Cancer du tiers moyen de l'œsophage *Acta gastro-entérol Belg*, Bruxelles 11 5 mai 1948 pp 1067-1078
- Mason (J. M.) Surgical treatment of obstructive lesions of the esophagus *Int Surg* 17, 5 mai 1948, pp 1067-1078
- Mes (G. M.) New method of esophagoplasty *J Internat Coll Surg* 1, mai juin 1948 pp 270-277
- Lortat Jacob Interventions pour cancer de l'œsophage *Arch des mal de l'app d g* 37, 5-6 mai juin 1948
- Maier (H.) Soins pre-opératoires opératoires et post opératoires dans les résections de l'œsophage *Surgery* 23, 6 juin 1948
- Pank (G. T.) Sherman (R. S.) Maier (H. C.) Watson (W. L.) Pool (J. L.) Garlock (J. H.) Payne (J. H.) Clagett (T.) De Amesti (F.), Otazu (E.) De Bakey (M.) Oschner (A.) and Sweet (R. H.) Revue générale sur le traitement de cancers de l'œsophage et du cardia *Surgery* 23 6, juin 1948
- De Bakey (M. E.) et Oschner (A.) Œsophagectomie subtotale et œsophagogastréctomie dans les lésions œsophagiennes intra thoraciques supérieures *Surgery* 23 6 juin 1948 pp 921-934
- Woodward (Fletcher D.) The surgical treatment of post cricoid and cervical esophageal carcinoma *Quart Rev Oto Rhino Laryngol and Broncho-Laryngol*, 7, 2, juin 1948
- Homburger (F.) Nutritional management of with transthoracic abdominal resection of the stomach *Surgery*, 23 juin 1948 p 1011
- Lefèvre (M. H.) Sur deux cas de cancers de l'œsophage guéris opératoirement Présentation des malades *Soc de chir de Bordeaux séance du 22 juil 1948*
- Garlock (J. H.) Résection de l'œsophage thoracique pour cancer situé au-dessus de la crosse de l'aorte *Surgery* 24 1 juil 1948 pp 1-8
- Macmanus (J. E.) Abord combiné abdominal et thoracique droit dans la résection de l'œsophage pour le cancer *Surgery* 24 1 juil 1948 pp 9-16
- Mouchet (A.) et Rouget (J.) Considérations sur le diagnostic et le traitement du cancer de l'œsophage thoracique *Ann d oto laryngol* 65 7, juil 1948
- Mouchet (A.) A propos du traitement chirurgical du cancer de l'œsophage thoracique *Arch des mal de l'app dig* 37 7-8 juil aout 1948 pp 518-523
- Rapant (Vladislav) et Kucera (Jan) (Olomouc) Œsophago-jéjunostomie et œsophago gastrotomie dans les cas de tumeurs inopérables de l'œsophage ou du cardia *Lekarske Listy* 3 16-17 15 aout 1948 pp 397-402
- De Bakey (M. E.) et Oschner (A.) Surgical treatment of malignant and benign obstructive lesions of the esophagus *Southern Surg* 14 aout 1948 pp 562-575
- Reynolds (J.) et Young Jr L'emploi de l'anastomose en Y de Roux pour étendre l'opérabilité du cancer de l'estomac et de la partie basse de l'œsophage *Surgery* 24 2 aout 1948
- Lions L'anesthésie en chirurgie thoracique en particulier de l'œsophage *New York State J Med* 48 17 1 sept 1948
- Sénéque (J.) A propos de l'œsophago gastro tomie dans le cancer inextirpable de l'œsophage *Arch des mal de l'app dig*, 37 9-10 sept-oct 1948 pp 599-601
- Nuboer Chirurgie du cancer de l'œsophage *Nederl tijdschr voor geneesk*, Haarlem 92, 41 9 oct 1948
- Mouchet (A.) Un an de chirurgie du cancer de l'œsophage *Acad de chir* 10 oct 1948
- Mouchet (A.) Etat actuel de la chirurgie du cancer de l'œsophage *XXI Congrès franç d O R I Paris* 18-23 oct 1948
- Kaiser (E.) (Wädenswil) Contribution à la résection transthoracique du cancer du tiers inférieur de l'œsophage *Schweiz med Wochenschr* no 42 23 oct 1948 pp 1035-1039
- Sénéque (J.) et Roux (M.) Œsophago-gastrostomie pour néoplasme cardio-œsophagien *Acad de chir* 10 nov 1948

- Resano (J. H.) Sur 100 œsophagectomies thoraciques pour néoplasie *Prensa med argent* 35 46 12 nov 1948
- Léger (L.) La pylorotomie ou pyloroplastie immédiate complément utile de l'œsophagectomie pour cancer avec rétablissement de la continuité *Soc nat franç de gastro Enterol* séance du 15 nov 1948
- Figarella (M.) Opération de Torek pour un cancer de l'œsophage thoracique *Soc de chir de Marseille* 22 nov 1948
- Dor (J.) et Spitalier Cancer de l'œsophage au tiers moyen œsophagectomie par voie droite *Soc de chir de Marseille* 29 nov 1948
- Campo (J.) Traitement chirurgical du cancer de l'œsophage thoracique *Med y Cir de Guerra Madrid* 10 11 nov 1948
- Senneville (A.) et Griffie Inscription graphique continue de la tension artérielle au cours d'une œsophagectomie *Soc de chir de Lille* séance du 10 déc 1948
- Monod (R.) et Hertzog Cancer de l'œsophage Œsophagectomie avec un nouveau procédé d'anastomose œsophago gastrique haute trans thoracique Résultats au bout de six mois *Acad de chir* 22 déc 1948
- Sweet (R.) Le carcinome du segment médiastinal supérieur de l'œsophage *Surgery* 24 6 déc 1948
- Bracco (R.) L'apport italien à la chirurgie du poumon et de l'œsophage *Bruxelles méd* 28 42 17 oct 1948 pp 2187-2189
- Magendie (J.) Le traitement chirurgical actuel du cancer de l'œsophage *Sud méd et Chir Marseille* a 79 2293 31 août *In J de chir* 64 1-2 1948
- Mouchet (A.) Trois malades porteurs de néoplasme de l'œsophage thoracique opérés par Resano et Cl Mouchet et guéris opératoirement *Acad de chir* 19 nov *In J de chir* 64 6 (55) p 36
- Hartl (H.) Anastomose gastro-œsophagienne *Der Krebsarzt* Vienne 3 2 1948 pp 25-26
- Resano (J. H.) Anatomie chirurgicale de l'œsophage thoracique *Presse méd* no 2 5 janv 1949
- Mer (P.) Sur la résection de l'extrémité inférieure de l'œsophage pour cancer du cardia *Ouest méd* Paris 7 1 10 janv 1949 pp 4-5
- Mouchet (A.) La chirurgie du cancer de l'œsophage thoracique *Ouest méd* Paris 2 1 10 janv 1949 pp 5-14
- Dor (J.) A propos du traitement chirurgical du cancer de l'œsophage moyen *Soc de chir de Marseille* séance des 15 et 16 janv 1949
- Decoux (M. P.) Decoux (Mme J.) et Senneville (A.) Œsophagectomie pour cancer par voie thoraco abdominale *Lille chir* 4 19 janv fév 1949 pp 8-17
- Dumont Ruyters et Crismer (R.) Diagnostic des tumeurs du pôle supérieur de l'estomac et de l'œsophage abdominal *Rev méd de Liège* 4 3 1 fév 1949
- Lanzara (A.) Intervention radicale par voie transthoracique dans deux cas d'adeno-carcinome de l'œsophage *Il Policlunco Sezione Pratica* Rome 56 III 7 fév 1949 pp 165-168
- Santy et Maillot Chirurgie du cancer de l'œsophage *Acad de chir* 23 fév 1949
- Chalnot (M.) A propos du traitement chirurgical du cancer du cardio-œsophage *Acad de chir* 27 avril 1949
- Mouchet (A.) et Mathis (Mme Ph.) Des anastomoses palliatives intra thoraciques dans le cancer de l'œsophage thoracique *Paris méd* 39 16 30 avril 1949
- Lefevre Cancer sus aortique de l'œsophage opéré par voie droite Transposition pre thoracique de tout l'estomac raccord par tube cutané *J de méd de Bordeaux et du Sud Ouest* 126 4 avril 1949
- Taylor (H.) Résultat éloigné d'une œsophagectomie suivie d'œsophago-gastrostomie pré sternale *Brit J Surg* 36 144 avril 1949 pp 419-421
- Santy (P.) et Maillot (P.) Cancer de l'œsophage cervical Exérèse suivie d'œsophago-gastrostomie *Soc de chir de Lyon* séance du 5 mai 1949 *In Lyon chir*
- Léger (L.) Voie d'abord de l'œsophage thoracique (pour la chirurgie du cancer) *Presse méd* no 33 21 mai 1949
- Lortat Jacob (L. L.) Voie d'abord de l'œsophage thoracique pour la chirurgie du cancer *Presse méd* 37 33 21 mai 1949
- Welti (H.) Cardio-œsophagostomie pour cancer inextirpable de l'œsophage *Acad de chir* 25 mai 1949
- Thompson (H. L.) Traitement chirurgical du carcinome de l'œsophage et de l'estomac *Arch Surg* 58 5 mai 1949 pp 662-678 *In West J Surg Obst and Gynec* 57 5 mai 1949 pp 205-209
- Froelich (F.) Hoffmann (R.) et Hubert (Ed.) Cancer du tiers inférieur de l'œsophage thora

- cique Résection œsophagienne Gastrectomie totale Anastomose œsophago-jéjunale *Strasbourg méd*, 109, 17, 15 juin 1949
- Decker (P) Le traitement opératoire du carcinome de l'œsophage *Schweiz. med. Wochenschr.* Bale, 79 24, 18 juin 1949 pp 535-537
- Olivier (Cl) L'anastomose palliative dans les cancers de la partie moyenne de l'œsophage thoracique *Presse méd* no 42 25 juin 1949
- Bricker (E M) Burford (T H) et Eisman (B) L'emploi des greffes cutanées pédiculées pour les œsophagoplasties intrathoraciques dans les cancers de l'œsophage supérieur *J Thoracic Surg* 18 3 juin 1949 pp 304-315
- Shefts (L M) et Fischer (A) Cancer de l'œsophage cervical Résection en un temps avec pharyngo-gastrostomie *Surgery*, 25 6 juin 1949 pp 849-861
- Lortat Jacob (J I) Enseignement d'une année de chirurgie du cancer de l'œsophage thoracique, étude analytique prévention et traitement des complications post opératoires, amélioration globale des résultats immédiats *Sem. des hôp. de Paris* 25 53 18 juil 1949, pp 2255-2267
- De Vido (G) (Padoue) Œsophagectomie pour carcinome de l'œsophage cervical Rapport de 5 cas *IV Congrès internat d'Oto Laryngol* Londres 18-23 juil 1949
- Antoine (M) et Massol (M) Radiologie du cancer de l'œsophage opéré *Sem. d'hôp. de Paris*, 25, 24 22 juil 1949
- Nissen (R) Œsophago-gastrostomie cervicale après résection d'un cancer sus-aortique de l'œsophage *Ann Surg* 130 1 juil 1949 pp 21-30
- Wylie (R H) et Frazell (E L) Anastomose œsophago-gastrique cervicale après résection de l'œsophage pour cancer *Ann Surg* 130 1 juil 1949
- Brewer (I A) Résection d'un cancer de l'œsophage cervical et œsophago-gastrostomie sous laryngée en un temps Un succès *Ann Surg* 130 1 juil 1949 pp 9-20
- Tanner (N C) Quelques considérations techniques sur la résection du cancer intéressant l'œsophage inférieur *J Internat Coll of Surgeons*, 12 4 juil août 1949
- Rudler (J C) Chirurgie des cancers de l'œsophage thoracique et du cardia *Lille chir* 4 22, juil août 1949
- Santy et Maillet (P) Chirurgie du cancer de l'œsophage *Lyon chir* 44 4 juil août 1949
- Brown (A L) Cancer de l'œsophage *J Thoracic Surg* St Louis 18 4 août 1949 pp 943-945
- Clark (D E) Sarcome de l'œsophage, résection avec succès d'un fibro-sarcome *Arch Surg*, 58 2 août 1949
- Dwight (E) Un cas de résection de fibro-sarcome du 1/3 supérieur de l'œsophage avec succès *Arch Surg*, 59 2 août 1949, pp 348-354
- Séry (Z) Notre tactique et technique opératoire dans le traitement des tumeurs malignes du cardia et de l'œsophage, et des cancers étendus de l'estomac *Lekarske Listy* Brno 4 17-18 1 sept 1949
- Goni Moreno (I) Résection de l'œsophage thoracique pour cancer *Prensa méd. argent* Buenos Aires 36 35 2 sept 1949
- Nissen (R) (New York) L'état actuel de l'opération radicale du cancer de l'œsophage thoracique *Deutsch. med. Wochenschr* 74 35 2 sept 1949 pp 1039-1041
- Pérez (R. Alb) La chirurgie dans le cancer de l'œsophage thoracique *Semana méd* Buenos Aires 56 36 8 sept 1949
- Stropecki (L) et Battezzati (M) (Genes) Gastro-œsophagostomie palliative dans les tumeurs œsophagiennes inopérables de siège cardiaque *Minerva chir*, 4, 18 15 sept 1949 pp 503-505
- Wilson (C P) Position actuelle au sujet du traitement chirurgical du cancer œsophagien *Royal Society of Medicine* no 18 oct 1949
- Rudler (J C) Cancer mœdithoracique de l'œsophage enlevé par thoracotomie droite première suite dans le même temps d'œsophagostomie au cou et d'œsophagoplastie antéthoracique avec l'estomac *Acad. de chir* 19 oct 1949
- Rob (C G) et Bateman (G H) La reconstruction de la trachée et de l'œsophage cervical observation préliminaire *Brit J Surg* 36 146 oct 1949
- McNeer (J) Les principes à observer dans les anastomoses de l'œsophage *Surgery* 26 4 oct 1949 pp 500-599
- Rudler (J C) Sur 66 interventions pour cancers de l'œsophage thoracique et du cardia *Maroc méd* oct 1949
- Lord (J W) Jr et Locahio (S A) Œsophago-gastrostomie cervicale haute pour cancer de l'œsophage cervical étendu au médiastin supérieur La valeur d'une voie d'approche thoraco-abdominale droite associée *New York State J Med* 49 21 1 nov 1949

- Kaufmann (M) et Nuloff (P) Résections œsophago gastriques *Canad Med Assoc J* 61 5, nov 1949
- Mouchet (A) Le traitement chirurgical du cancer de l'œsophage thoracique indications techniques résultats *Sem des hop* 25 91, 10 déc 1949
- d'Allaines et Banzet Etude du traitement du cancer de l'œsophage *Réunions chir de l'hop Broussais* 12 déc 1949 in *Sem des hop* III mai 1950
- Rudler (J C) L'exérèse des cancers haut situés de l'œsophage thoracique par thoracotomie droite première suivie d'œsophagoplastie antéthoracique avec l'estomac *Presse méd* 37 81 25 déc 1949
- Lortat Jacob (J L) Les indications du traitement chirurgical du cancer de l'œsophage thoracique *Gaz méd de France* 57 24 25 déc 1949
- Lortat Jacob (J L) et Robert (H G) Résultats statistiques *Gaz méd de France* 57 24 25 déc, 1949
- Le Brigand (J) et Brunet D Aubiac (P) Complications et soins pré per et post-opératoires *Gaz méd de France* 57 24 25 déc 1949
- D Allaines (F) Dubost (C) et Gallez (J J) Œsophago gastrostomies palliatives sans résection dans les cancers de l'œsophage et du cardia *J de chir* 65 6 1949 pp 289-301
- Mathis Foisy (Mme S) A propos de l'anastomose palliative sus tumorale dans les cancers inextirpables de l'œsophage moyen Indications et technique *Thèse Paris* 1949
- Hilgenfeldt (O) Sur le traitement opératoire du cancer de l'œsophage inférieur et de la partie supérieure de l'estomac *Zentralbl fur Chir* Leipzig 74 9 1949
- Harrington (S W) Traitement chirurgical des tumeurs bénignes puis des tumeurs malignes de l'œsophage *Arch Surg* 58 5 1949 pp 646-661
- Staudacher (V) Bellinazzo (P) et Belli (L) (Padoue) Contribution à l'étude des anastomoses termino-terminales de l'œsophage *Chirurgie* 4 3 1949 pp 197-208
- Halligan (E J) Perkel (L) et Catlav (J K) Le traitement chirurgical du cancer de l'œsophage *Am J Surg* 79 2 fév 1950
- O'Toole (P) L'anesthésie pour cancer de l'œsophage *Irish J Med Sc* Dublin 6 290 fév 1950
- Scott (H W) Jr et Hanlon (C R) Voies d'approche cervicale et thoracique simultanées pour la résection du quart supérieur de l'œsophage un cas avec anastomose œsophago gastrique *Ann Surg* 131 2 fév 1950
- Trojanowski Cancer chirurgie *J de chir* 66 2 fév 1950
- Mouchet (A) L'anastomose palliative intra thoracique dans les néoplasmes de l'étage moyen *Soc nat franç de gastro entéro* 13 mars 1950
- Perrotin (J) Technique de l'œsophagectomie transpleurale gauche *J de chir* 66 4 avril 1950
- Aksel (A) Acquisitions récentes dans la chirurgie de l'œsophage *Türk Tip Cemiyeti Mecmuası* Istanbul 16 1 1950
- Nemours Auguste et Raff Un cas de cancer du tiers moyen de l'œsophage opéré *Soc nat franç de gastro entéro* 15 mai 1950
- Santy (P) et Michaud (P) Œsophagectomie thoracique totale pour cancer haut situé de l'œsophage thoracique Anastomose œso-gastrique au cou *Lyon chir* 45 8 nov déc 1948
- Resano (J H) Ciento cincuenta y cinco sobrevividas a esofagectomía por neoplasia del esófago (One hundred and fifty five cases of survival after esophagectomy for cancer) *Prensa med argent* Buenos Aires 37(17) 866-873 1950
- Brown (R) A two-rib incision for a subtotal esophagectomy *Ann Surg* 131(4) 588-591 1950
- Reynolds (J T) Holinger (P H) Andrews (A H Jr) Young (J P Jr) and Marlowe (W H) Role of tracheotomy in the postoperative care of patients subjected to esophagectomy *Arch Surg* 61(2) 211-228 1950
- Nuboer (J F) Enkele beschouwingen over de chirurgische behandeling van carcinoma oesophagi (Some considerations on the surgical treatment of cancer of the esophagus) *Ned tijdschr geneesk* 4(44) 3203-3206 1950
- Santy (P) and Michaud (P) Position actuelle et possibilités de la chirurgie du cancer de l'œsophage thoracique conclusions tirées de l'observation de plus de 300 cas en 3 ans (Present position and possibilities of surgery for carcinoma of the thoracic esophagus conclusions drawn from the study of over 300 cases within 3 years) *Arch mal appar dig* 40(7-8) 932-940 1951
- Resano (J H) Resección del cáncer de esófago supraaorticocon anastomosis esófagogástrica cervical (Resection for carcinoma of the supra aortic esophagus with cervical esophago-gastrostomy) *Prensa m d argent* 38(36) 2287-2295 1951

- Nuboor (J F) Chirurgische mogelijkheden bij de behandeling van sloot darmkanker (Surgical possibilities in the treatment of cancer of the esophagus) *Ned tijdschr geneesk*, 96,35) 2171-2175 1952
- Sweet (R H) The results of radical surgical extirpation in the treatment of carcinoma of the esophagus and cardia With five year survival statistics *Surg Gynec & Obst* 94,1) 46-52 1952
- Breerema (I) Oesophagus resection with restoration of continuity by a gastric tube *Arch chirurg* 4,2) 120-130 1952
- MacJeter (S A) and Mayer (R M) Palliation of esophageal obstruction due to carcinoma with a permanent intraluminal tube *J Thoracic Surg* 28,4) 431-445 1954
- Sweet (R H) Present status of the treatment of carcinoma of the esophagus *Arch Surg* 69,1) 1-3 1954
- Sweet (R H) Late results of surgical treatment of carcinoma of the esophagus *J A M A* 155,5) 422-425 1954
- Nakayama (K) Approach to midthoracic esophageal carcinoma for its radical surgical treatment *Surgery* 35,4) 574-589 1954
- Rambert (R) Jouanneau (P) and Gessroy (Y) L'intubation métallique des cancers de l'œsophage inopérables (technique de Resano) (Insertion of metal tubes in cases of inoperable cancer of the esophagus [Resano's technique]) *Arch mal appar dig* 43,11) 1058-1061 1954
- Raven (R W) The surgical treatment of carcinoma of the hypopharynx *Brit J Surg*, 42,172) 113-122 1954
- Garlock (J H) and Klein (S H) The surgical treatment of carcinoma of the esophagus and cardia (an analysis of 457 cases) *Ann Surg* 139,1) 19-34 1954
- Garlock (J) Résultats à distance du traitement chirurgical du cancer de l'œsophage et du cardia (Late results of surgical treatment of cancer of esophagus and cardia) *Mem Acad chir* (Paris) 80,23-24) 686-690 1954
- Adams, W L) The future outlook of surgical therapy for carcinoma of the esophagus *Surg, Gynec & Obst* 100,3) 366-368 1955
- Grimes (O F) and Stephens (H H) The treatment of carcinoma of the hypopharynx and cervical esophagus *Arch Surg* 77,5) 742-745 1956
- Sweet (R H) End results in cancer of the esophagus *Proc Third National Cancer Conference* J B Lippincott Co Philadelphia 1956
- Heimlich (H J) Greenlees (T W) and Winfield (J M) A palliative procedure with use of a plastic tube introduced through a cervical incision for carcinoma of the esophagus *J A M A* 161,3) 192-194 1956

Chapter 29 - Irradiation Treatment of Carcinoma

- Jacobson (M) Cancer de l'œsophage encore en vie six ans après radiothérapie *Acta radiol*, mai 1946 pp 331-357
- Buttkofer L Irradiation rotatoire du cancer de l'œsophage *Radiol clin Bâle* 16 1 1947 pp 7-12
- Buschke (F) et Cantyl (S T) La radiothérapie à très haut voltage du cancer de l'œsophage *J A M A* 134 2 10 mai 1947, pp 127-130
- Heming (J A C) La radiothérapie du cancer de l'œsophage thoracique *Thorax*, 2 4 déc 1947 pp 206-216
- Paterson (R) *The treatment of malignant disease by radium and X-rays* Edward Arnold et Cie Londres 1948
- Lefèvre (L) Le traitement radiothérapique du cancer de l'œsophage *Paris méd* 20 mars 1948 pp 142-146
- Freid (J R) Traitement radiothérapique du cancer de l'œsophage *Am J Roent and Rad* 59 4 avril 1948 pp 551-564
- Moulonguet et Mallet La cyclothérapie dans les cancers œsophagiens *Acad de chir*, séance du 17 nov 1948
- Piquet (M J) Un cas de tumeur sténosante de l'œsophage thoracique traité par radiothérapie et guéri après cinq ans *Soc de chir de Lille* séance du 10 déc 1948
- Krebs (C) Nielsen (Howard) et Andersen (P E) La cyclothérapie du cancer de l'œsophage *Acta Radiol* 37 4 189 31 oct 1949 pp 304-310
- Surmont (J) Markovits (P) et Gest (J) Radiothérapie du cancer de l'œsophage *Gaz. méd de France* 57 24 déc 1949 pp 937-938
- Baclesse Reverté (J) Jamet (H) et Desclaux (J) Etude du traitement du cancer de l'œso-

phage Réunions chirurgicales de l'Hôpital Broussais 12 décembre 1949 *Sem d hop de Paris* 18 mai 1950, p 1742

Frimann Dahl (J) Rotation therapy in cancer of the esophagus *Acta chir scand* 103(6) 421-424 1952

Kropff (G) Joinville (R) and Legrand (J) Intubation œsophagienne par tube en matière plastique suivie de radiumthérapie dans le traitement du cancer de l'œsophage (Introduction of a tube of plastic material into the esophagus followed by radium therapy in the treatment of cancer of the esophagus) *Gaz med France* 61(17) 993-1007 1954

INDEX

Italic folios indicate pages with illustrations

- ABSCCESS(ES)**
mediastinal 315
parietal localized 309
characteristics of 309
prognosis in 309
treatment of 309
periesophageal carcinoma of esophagus and 506
- Acanthosis nigricans** of esophagus 353
- Achalasia** *See* Mega-esophagus
- Actinomycosis** of esophagus 349
carcinoma of esophagus vs 533
characteristics of
clinical 350
endoscopic 350
pathology of 349
prognosis in 350
treatment in 350
- Adenopathy** carcinoma of esophagus and 527
- Adrenalin** deglutition and 48
- Aerophagia** 67
in mega esophagus type 1 210
- Alcoholism** malignant tumors of epithelial origin and 503
- Ampulla** of esophagus 73
phrenic 44 45 46
- Anastomosis**
portacaval for esophageal vances 287
technique of 289
splenorenal for esophageal vances 287
technique of 288
- Anatomy** of esophagus 1-42
gross 1-38
microscopic 38-42
- Antibiotics** after esophagectomy with esophagogastric anastomosis 552
- Antrum** cardinal roentgen appearance of 61
- Aorta** erosion of carcinoma of esophagus and 506
- Arteria lusoria** 163
- Artery**
Gossart's 23
Luschka's esophageal 21
thyroid inferior sympathetic plexus of 22
- Aspiration** bronchoscopic after esophagectomy 566
- Atelectasis** pulmonary after thoracic esophagectomy 566
- Atony** of esophagus *See* Paralysis of esophagus
- Atropine** deglutition and 47
- Auerbach's plexus** 37
- BANTHINE** deglutition and, 47
- Barsony Teschendorff syndrome** 11
characteristics of
clinical 184
endoscopic 187
roentgen 185
diagnosis in differential 187
etiology of 183
pathogenesis of 183
prognosis in 188
treatment in 188
- Biopsy** esophageal 113
in carcinoma of esophagus 113
- Bleeding** *See also* Hematemesis and Hemorrhage(s)
from esophagus 276
- Bougienage** *See also* Esophageal dilatation
mental
devices for 226
in acute corrosive esophagitis 11
in Barsony Teschendorff syndrome 11
in mega-esophagus 22
in peptic ulcer of esophagus 11
in spasm of pharynx 176

- Bougienage perforation of esophagus during 444
- Brachyoesophagus 145
- characteristics of
 - clinical
 - in adults 149
 - in children 147
 - endoscopic
 - in adults 149
 - in children 149
 - roentgen
 - in adults 149
 - in children 147
 - diagnosis in differential 150
 - etiology of, 146
 - Marfan's syndrome and, 146
 - pathogenesis of 146
 - peptic ulcer of esophagus and 323
 - prognosis in 151
 - treatment of 151
- Bronchoscopy in congenital malformations of esophagus and trachea 135
- Bronchus (Bronchi) lesions of acquired esophagorespiratory communications and 423
- Burns chemical of esophagus See Esophagitis, corrosive acute
- CACHEXIA** carcinoma of esophagus and 516
- Canal diaphragmatic See Hiatus esophageal
- Carcinoma See also Tumors, malignant
- of cardia treatment of, surgical end results of 571
 - of esophagus
 - acquired esophagorespiratory communications and 424
 - cardiovascular complications and 528
 - characteristics of
 - clinical 514 529
 - at onset 514
 - during development, 516
 - terminal 521
 - endoscopic, 531
 - at onset 515
 - during development 518
 - terminal 521
 - roentgen 531
 - at onset 515
 - during development, 517
 - terminal 521
 - complications of, 526
 - involving diaphragm, 528
 - involving nervous system 528
 - course of clinical abnormalities of 523
 - cytology in exfoliative 532
 - diagnosis of 529
 - diagnosis in differential 532
 - epidermoid 504
 - characteristics of physical 505
 - location of, 504
 - size of 505
 - Carcinoma of esophagus, erosion of aorta and 506
 - extrinsic lesions vs 532
 - intrinsic non malignant lesions vs 533
 - location of, treatment and surgical 536
 - mega-esophagus and 220 223
 - midmediastinal treatment of surgical 545
 - nutrition in diets for maintaining 591-595
 - of cervical segment
 - treatment of surgical end results of, 569
 - Wookey procedure for 556
 - complications after 563
 - first stage, 556
 - second stage 560
 - of lower segment
 - treatment of, surgical end results of, 571
 - technique of 537
 - alternative, 543
 - of superior mediastinal segment treatment of surgical end results of 570
 - of thoracic segment
 - treatment of surgical end results of 570
 - technique of 537
 - periesophageal abscess and 506
 - recurrent laryngeal nerve involvement and 529
 - signs of objective 517
 - special considerations in 523
 - treatment of
 - irradiation, 575-590
 - difficulties of 575
 - radium, 576
 - end results of 582
 - roentgen 583
 - adjuvant treatment with 588
 - complications of 587
 - dosages in calculation of 583
 - end results of 589
 - frequency of 586
 - indications for, 586
 - palliative 587
 - radical 586
 - techniques of 584
 - surgical 535-574
 - end results of, 569
 - objective of 535
 - palliative 572
 - variations in
 - anatomical types and 524
 - location and 524
 - of other organs
 - invasion of esophagus by 503
 - metastatic to esophagus 503
- Cardia
- paralysis of 242
 - passage through 76
 - roentgen examination of 61
- Cardioplasty in mega-esophagus, 228

- Cardiospasm *See* Mega-esophagus
- Cardiovascular system complications involving carcinoma of esophagus and 528
- Cobalt bomb irradiation by 590
- Colon use of for esophageal replacement 413
- Communications esophagorespiratory acquired 419-434
 characteristics of
 clinical 427
 endoscopic 430
 physical 424
 roentgen 429
 diagnosis in differential 430
 etiology of 420
 lesions associated with 426
 pathogenesis of 424
 pathology of 424
 prognosis in 432
 treatment in 432
- Compression of esophagus
 by acquired abnormalities of aorta 171
 characteristics of
 clinical 171
 endoscopic 172
 roentgen 172
 treatment of 172
 by congenital vascular malformations 163
 characteristics of
 clinical 167
 roentgen 168
 embryology of 164
 treatment of 170
 localized 160
 treatment of 163
- Coughing foreign bodies and 459
- Cricopharyngeus: muscle: sphincter action of 8
- Curetherapy 576
- Curling of esophagus 45 181
 characteristics of
 endoscopic 182
 roentgen 181
 etiology of 181
 treatment of 182
- Cyst(s) of esophagus 491
 characteristics of histological 491
 congenital 152
- Cytology exfoliative of esophagus 114
 in carcinoma of esophagus 532
- DEGLUTITION
 center for 47
 control of nervous 47
 difficult foreign bodies and 459
 fluoroscopic observation of 70
 of liquids 71
 in infants 79
 of pastes 71
 of solids 70
 phases of 43
 stimuli and noxious 46
- Diaphragm complications involving carcinoma of esophagus and 528
- Diets for patients with dysphagia 591-595
- Digestion in esophagus 50
- Dilatation of esophagus
 carcinoma and 506
 congenital *See* Mega-esophagus
 idiopathic *See* Mega-esophagus
- Dilator(s)
 for use in mega esophagus 226 227 228
 hydrostatic 228
 Starck 228
- Diphtheria of esophagus 347
 characteristics of clinical 347
 diagnosis in differential 348
 etiology of 347
 prognosis in 348
 treatment in 348
- Diverticulum (Diverticula) 247-275
 definition of 247
 epiphrenic *See* Diverticulum of lower esophagus
 juxtaspincteric 247
 mechanism of 248
 of lower esophagus 264
 characteristics of
 clinical 265
 endoscopic 266
 roentgen 266
 complications of 268
 diagnosis in differential 267
 prognosis in 268
 treatment of 268
 surgical 268
 aftercare in 269
 complications of 271
 of pharyngo-esophageal segment 249
 characteristics of
 clinical 253
 endoscopic 255
 roentgen 254
 complications of 256
 diagnosis in differential 256
 prognosis in 257
 treatment of 257
 nonsurgical 257
 surgical 258
 aftercare following 262
 complications of 263
- pulsion 247
 mega-esophagus and 221
 site of 6
 traction *See* Pseudodiverticula
 types of 247
- Drainage after esophagectomy with esophago-gastric anastomosis 553
- Dysphagia
 carcinoma of esophagus and 516
 constriction of esophagus and subdiaphragmatic 5
 foreign bodies and 460
 in mediastinitis 316
 in mega-esophagus type I 210

- 674
- Dysphagia in paralysis of esophagus 237
 intermittent in infants 195
 characteristics of
 clinical 196
 endoscopic, 196
 roentgen, 196
 diagnosis in, differential, 196
 obstructive
 diets for patients with 591-595
 in peptic ulcer of esophagus, 329
 paradoxical, in mega-esophagus type 1, 211
 progressive, carcinoma of esophagus and
 529
 sideropenic *See* Esophagitis nonspecific
 chronic, of mouth of esophagus
 Dysphagia lusoria, 163
- EDEMA
 angioneurotic of esophagus 353
 pulmonary, after thoracic esophagectomy
 567
 Emaciation, carcinoma of esophagus and 516
 Empyema, after thoracic esophagectomy 567
 Endocurietherapy 576
 applicators for
 flexible, 579
 semirigid, 579
 complications of, 582
 contraindications to 582
 dosage in 581
 Guisez's method of, 579
 Sargnon's method of, 579
 with esophagoscopy, 577
 without esophagoscopy, 579
 Epithelioma of esophagus
 cylindrical, 509
 typical 508
 Esophageal varices *See* Varices
 Esophagectomy
 cervical
 complications after 563
 mortality after 564
 Wookey procedure for 556
 complications after 563
 first stage 556
 aftercare following 560
 mortality after 564
 second stage 560
 end results of 569
 partial with high intrathoracic esopha-
 gogastric anastomosis 545
 subtotal with intracervical esophagogas-
 tric anastomosis 550
 thoracic
 complications after 564
 due to errors of technique 564
 pulmonary 566
 mortality after 567
 with esophagogastric anastomosis
 aftercare following 552
 management of late recovery period
 555
- Esophagitis
 agranulocytic 295
 carcinoma of esophagus and 526
 carcinoma of esophagus vs., 533
 catarrhal *See* Esophagitis, nonspecific
 acute
 corrosive, acute, 358-375
 bougenage in 370
 characteristics of
 clinical, 365
 endoscopic 367
 roentgen, 365
 complications in 367
 diagnosis in differential 368
 etiology of, 358
 pathology of, 359
 macroscopic 361
 microscopic 362
 prognosis in, 368
 treatment in, 369
 results of, 375
 definition of, 292
 malignant tumors of epithelial origin
 and 503
 mega esophagus and 220
 nonspecific 292-307
 acute 293
 characteristics of
 clinical, 293
 endoscopic 294
 treatment of, 295
 types of, 293
 chronic 296
 characteristics of
 clinical, 298
 endoscopic 300
 roentgen, 299
 diagnosis in differential 300
 etiology of 296
 of mouth of esophagus 301
 characteristics of
 clinical 303
 endoscopic 305
 roentgen 304
 complications of 306
 diagnosis in differential 305
 etiology of 302
 evolution of 306
 pathogenesis of 302
 pathology of 303
 treatment of 306
 pathology of 297
 treatment of 300
 pain in, 50
 phlegmonous *See also* Phlegmon(s) of
 esophagus
 diffuse 314
 characteristics of clinical 314
 pathology of 314
 pyrosis and 50
 reflux esophagogastric junction and 21
 Esophagomyotomy
 in Barsony-Teschendorf syndrome 189

- Esophagomyotomy in mega esophagus, 229
 aftercare following 230
 results of 230
 technique of 229
- Esophagoplasty
 in mega esophagus 232
 aftercare following 233
 results of 233
 technique of 232
- Esophagoscope
 Haslinger 83
 Jackson use of 101
- Esophagoscopy 80-113
 accidents in 109
 prevention of 111
 treatment of 112
 anesthesia for 81
 contraindications to 80
 endocureththerapy using 577
 examination of patient prior to 81
 in acquired esophagorespiratory communi-
 cations 430
 in actinomycosis of esophagus 350
 in acute corrosive esophagus 367
 in Barsony Teschendorff syndrome 187
 in benign tumors of esophagus 499
 in brachyoesophagus
 in adults 149
 in children 149
 in carcinoma of esophagus 531
 developing 517
 early 515
 terminal 521
 in chronic nonspecific esophagitis 300
 of mouth of esophagus 305
 in chronic spasm of cricopharyngeus mus-
 cle 193
 in cicatricial stenosis 382
 in compression of esophagus by acquired
 abnormalities of the aorta 172
 in congenital malformations of esophagus
 and trachea 135
 in curling of esophagus 182
 in diverticulum
 of lower esophageal segment 266
 of pharyngo esophageal segment 255
 in esophageal varices 283
 in esophagospasm in children 198
 in foreign bodies in esophagus 465
 in injuries to cervical esophagus 440
 in instrumental perforation of esophagus
 451
 in intermittent dysphagia in infants 196
 in kyphoscoliosis 100
 in kyphosis 100
 in localized parietal abscesses 309
 in mediastinal abscess 317
 in mega-esophagus 218 223 224
 in nonsphincteric pseudodiverticula 275
 in paralysis of esophagus 243
 in partial stenosis of esophagus 128
 in peptic ulcer of esophagus 326
 in phlegmons of esophagus 312
- Esophagoscopy in scleroderma 356
 in spasm of epicardial segment 179
 in spasm of pharyngo esophageal segment
 176 177
 in syphilis of esophagus 345
 in tuberculosis of esophagus 340
 indications for 80
 instrumentarium for 83
 perforation of esophagus during 445
 preparation of patient for 111
 retrograde 107
 in cicatricial stenosis 389
 techniques of 85
 Haslinger 81
 with Chevalier Jackson esophagoscope
 101
- Esophagospasm 173-198
 acute 174
 etiology of 174
 of epicardial segment 177
 characteristics of
 clinical 177
 endoscopic 179
 roentgen 177
 diagnosis in differential 179
 prevention of 180
 prognosis in 180
 treatment in 180
 of pharyngo-esophageal segment 175
 characteristics of
 clinical 175
 endoscopic 176
 prognosis in 176
 carcinoma of esophagus vs 533
 chronic 189
 at cricopharyngeus muscle 189
 characteristics of
 clinical 192
 endoscopic 193
 roentgen 193
 etiology of 189
 pathogenesis of 191
 treatment of 195
 of epicardial segment 193
 characteristics of
 clinical 194
 roentgen 194
 treatment of 195
 in children 197
 characteristics of
 clinical 197
 endoscopic 198
 roentgen 198
 diagnosis in differential 198
 treatment in 198
 in infants 195
 of body of esophagus 181
 pyrosis and 50
- Esophagotomy
 cervical
 for extraction of foreign bodies 486
 aftercare in 488
 technique of 487

- Esophagotomy, thoracic, for extraction of
 foreign bodies 488
 transabdominal, for extraction of foreign
 bodies, 489
- Esophagus
 - abdominal perforation of, instrumental,
 surgery for, 454
 - abnormal endoscopic views of, 251
 - abscess of See Abscess of esophagus
 - activity of sensory 49
 - anatomy of, 1-42
 - gross, 1-38
 - microscopic 38-42
 - atony of See Paralysis of esophagus
 - biopsy of, 113
 - in carcinoma of esophagus 520
 - bleeding from, 276
 - blood supply of, 21-25
 - caliber of 4
 - characteristics of, general, 2-5
 - chorion of, 38
 - congenital short See Brachyoesophagus
 - constriction(s) of 4
 - aortic, 5
 - bronchial 5
 - cardiac, 5
 - cricopharyngeal 4
 - diaphragmatic 5
 - retrocricoid 7
 - retrosternal 5
 - subdiaphragmatic 5
 - covering of 21
 - curling of See Curling of esophagus
 - definition of 1
 - deviation of See under Position of esoph-
 agus
 - digestion in 50
 - direction of 2
 - diseases of associated with diseases of
 skin and mucous membranes 352-357
 - drainage of
 - lymphatic 27-28
 - venous 25-27
 - embryonal development of 123
 - epithelium of 38
 - exfoliative cytology of 114
 - in carcinoma of esophagus 532
 - foreign bodies in See Foreign bodies
 - function of 43
 - glands of 39
 - function of 50
 - impulses and extrinsic 46
 - in adult roentgen appearance of 54
 - in infant roentgen appearance of 67
 - inflammation of See also Esophagitis
 - pyogenic 308-319
 - etiology of 308
 - injuries to external 438-442
 - innervation of 39-38
 - extrinsic 30
 - intrinsic 37
 - sensory intrinsic 38
 - sympathetic 33-34
- Esophagus layers of 38
 - length of 3
 - lesions of acquired esophagorespiratory
 communications and 420
 - limits of, 1
 - mouth of, 5
 - deglutition and, 43
 - movements of
 - circulatory 96
 - respiratory, 95
 - mucous membrane of 20 38
 - folds of roentgen appearance of 66
 - muscular layer of 40
 - muscularis mucosae of 39
 - normal
 - endoscopic appearance of 108
 - roentgen appearance of
 - in adult 54
 - in infant, 67
 - obstruction of
 - carcinoma and, 526
 - chronic carcinoma and 503
 - pain in 49
 - paralysis of See Paralysis of esophagus
 - perforations of
 - carcinoma and 506
 - instrumental 443-455
 - characteristics of
 - clinical 446
 - endoscopic, 451
 - roentgen 449
 - etiology of 443
 - pathogenesis of 443
 - pathology of 445
 - postoperative care in 455
 - prognosis in 451
 - signs of
 - general 448
 - local 447
 - symptoms of, 446
 - treatment in 451
 - surgical 453
 - peristalsis in 44
 - physiology of, 43-51
 - reflexes and distant 50
 - rest of in instrumental perforation 457
 - role of in deglutition 43
 - rupture of, 435-438
 - spontaneous, 435
 - aftercare in, 438
 - characteristics of
 - clinical 436
 - physical 436
 - diagnosis in, differential 437
 - mechanism of 435
 - prognosis in 437
 - treatment in 437
 - traumatic 435
 - segment(s) of, 5-21
 - abdominal 20
 - blood supply of 24
 - innervation of 34
 - limits of 20

- Esophagus segment(s) of : abdominal relations of 21
 cervical 8
 blood supply of 21
 carcinoma of treatment of surgical 536
 extraction of foreign bodies from 484
 injuries to 438
 aftercare in 441
 characteristics of clinical 439
 complications of 440
 diagnosis in differential 439
 prognosis in 440
 treatment in 440
 innervation of 30
 sympathetic 31
 limits of 8
 perforation of instrumental surgery for 453
 relations of 11 12
 venous drainage of 25
 diaphragmatic 16
 lower carcinoma of treatment of surgical 536
 alternative 543
 midmediastinal carcinoma of treatment of surgical 545
 pharyngo-esophageal 5
 innervation of 30
 muscle fibers of 6
 weak points of 6 8
 superior mediastinal carcinoma of treatment of surgical 536
 thoracic 10
 blood supply of 22
 carcinoma of treatment of surgical 537
 injuries to 441
 characteristics of clinical 441
 diagnosis in differential 441
 prognosis in 441
 treatment in 442
 innervation of 33
 limits of 10
 perforation of instrumental surgery for 454
 relations of 13 14 15 16
 sub bronchial portion of 11
 suprabronchial portion of 10
 venous drainage of 25
 shape of 4
 spasm of *See* Esophagospasm
 sphincter of 8
 submucosa of 40
 substitution for 397-418
 colon for 413
 results of 416
 jejunum for 406
 technique of
 one stage 407
 two-stage 411
 methods of 397
 principles of general 397
- Esophagus substitution for skin tube for 397
 antethoracic 398
 cervical 398
 stomach for 401
 tube prosthesis for 417
 viscera of alimentary tract for 400
 routes of 400
 temperature sense in 50
 tortuosities of 3
 tube prosthesis for 417
 tumors of *See under* Tumors
 tunica propria of 38
 ulceration of *See also under* Ulceration(s)
 and Ulcer(s)
 pain in 50
 venous drainage of 25-27
- Exploration of esophagus instrumental 117-122
 bougies for 119
 complications of 122
 general considerations in 118
 in intermittent dysphagia in infants 196
 in mega-esophagus 217
 in paralysis of esophagus 243
 sedation for 120
 technique of 119
- FASCIA Laimer's 18 19
 Fever foreign bodies and persistent 460
 Fiber(s)
 Juvara's 17 18 19
 Rouget's 17 18 19
 Fibroma(ta) of esophagus 493
 Fistula(e) *following Esophagogastric fistula P 564*
 complicating carcinoma of esophagus 527
 esophagobronchial acquired *See* Communications : esophagorespiratory acquired
 esophagotracheal acquired *See* Communications : esophagorespiratory acquired
 Food passage of through esophagus 43
 Foreign bodies in esophagus 456-468
 carcinoma of esophagus vs 533
 characteristics of
 clinical 458
 endoscopic 465
 roentgen 461
 complicated extraction of procedures for 485
 complications of 468
 considerations in general 456
 diagnosis in differential 466
 extraction of 469-489
 anesthesia for 471
 from mouth of esophagus 484
 perforation of esophagus during 443
 technique of 470
- hard
 extraction of 472
 flat extraction of 473
 irregularly shaped extraction of 475

- Foreign bodies in esophagus hard regularly shaped extraction of 473
 round extraction of, 475
 with perforating points, extraction of 476
 with traumatizing angles extraction of 476
 metallic, extraction of, with jointed magnets, 482
 technique of 484
 persistent, symptoms of, 460
 problems in, special 479
 prognosis in, 468
 radiolucent, 463
 radiopaque, 461
 roentgen detection of 461
 signs in, physical 461
 soft, extraction of, 471
 symptoms of, 459
 early, 459
- GASTROSCOPY, perforation of esophagus during 445
- Gastrostomy
 in cicatricial stenosis 389
 in roentgen treatment of carcinoma of esophagus 588
- Globus hystericus 46, 223 303
- Gossart's artery 23
- Guisez's method of endocunethery, 579
- HALITOSIS, carcinoma of esophagus and 516 531
- Haslinger esophagoscope 83
- Haslinger head rest for esophagoscopy, 85
- Haslinger technique of esophagoscopy, 85
- Headrest, Haslinger, for esophagoscopy 85
- Heart, complications involving after thoracic esophagectomy, 567
- Heartburn, 40
- Heller operation in Barsony-Teschendorff syndrome 189
- Hemangioma(ta) of esophagus, 291, 497
- Hemangiomatosis familial hereditary, 291
- Hematemesis, foreign bodies and persistent 460
- Hemorrhage(s) *See also* Bleeding and Hematemesis
 cerebral after thoracic esophagectomy 567
 in peptic ulcer of esophagus 325
- Herpes of esophagus 353
- Hiatus esophageal, 16 17 18
 fascia of 18 20
- Hypertrophy giant muscular *See* Esophagospasm chronic of epicardial segment
- INFECTIONS of esophagus *See also* Esophagitis
 mycotic 349
 prophylaxis of in instrumental perforation of esophagus 452
 specific 333
- Inflammation of esophagus *See* Esophagitis and under Esophagus
- JACKSON esophagoscope, esophagoscopy with, 101
- Jackson sign, 461
- Jackson speculum 102
- Jejunum use of for esophageal substitution 406
- Juvara's fibers 17, 18, 19
- KYPHOSCOLIOSIS
 deviation of esophagus and 156
 esophagoscopy in, 100
- Kyphosis esophagoscopy in 100
- LARIMER'S fascia 18, 19
- Larynx, complications involving carcinoma of esophagus and, 527
- Leucoplakia mega esophagus and 220
- Ligament phreno-esophageal 19
- Lipoma(ta) of esophagus, 496
- Luschka's esophageal artery, 21
- Lymphangioma(ta) of esophagus, 497
- Lymphatic drainage of esophagus, 27-28
- Lymphogranulomatosis of esophagus, carcinoma of esophagus is 533
- MAGNETS jointed, for extraction of metallic foreign bodies, 482
- Malformations congenital 123-153
 of esophagus alone 124
 of esophagus and trachea, 129
 bronchoscopy in 135
 characteristics of
 clinical 132
 endoscopic 135
 roentgen 133
 pathogenesis of 132
 treatment of, 135
 surgical, 135
 anesthesia for 136
 contraindications to, 135
 one stage procedure 140
 preparation for, 136
 primary anastomosis in 140
 technique of 137
 two-stage procedure 143
- Marfan's syndrome brachy-esophagus and 146
- Mechols' deglutition and 47
- Medastinitis 315
 carcinoma of esophagus and 526
 characteristics of
 clinical 316
 roentgen 316
 diagnosis in differential 317
 pathology of 315
 prognosis in, 317
 treatment in 318
 results of, 319

- Mediastinotomy**
 cervical in mediastinitis 318
 thoracic in mediastinitis 318
Mediastinum
 infections of *See* Mediastinitis
 lesions of acquired esophagorespiratory
 communications and 422
Mega-esophagus 199-235
 characteristics of
 clinical 208
 endoscopic 218
 histological 204
 complications of 220
 regional 222
 diagnosis in differential 222
 dietary measures in 224
 etiology of 199
 exploration in instrumental 217
 pathogenesis of 201
 treatment of 224
 mechanical 225
 medical 225
 surgical 228
 types of 208
 1 209
 characteristics of roentgen 213
 symptoms of 209
 2 215
 characteristics of roentgen 216
Meissner's plexus 37
Meticorten in roentgen treatment of car-
 cinoma of esophagus 588
Minnigerode sign in instrumental perfora-
 tion of the esophagus 449
Mucosa of esophagus 38
 at esophagogastric junction 20
 folds of roentgen appearance of 66
Muscle cricopharyngeus sphincter action
 of 8
Myoma(ta) of esophagus 494
Myxoma(ta) of esophagus 497
Myxosarcoma(ta) of esophagus 513

NEOPLASIA *See also* Tumors
 mega-esophagus and 220
Nerve recurrent laryngeal involvement of
 carcinoma of esophagus and 529
Nervous system
 complications involving carcinoma of
 esophagus and 528
 extrinsic of esophagus 30
 intrinsic of esophagus 37
Nielson's method of roentgen therapy of
 carcinoma of esophagus 585
Node(s) lymph of esophagus 27 28
Nutrition
 after esophagectomy with esophagogastric
 anastomosis 554
 in carcinoma of esophagus diets for main-
 taining 591-595
 in roentgen treatment of carcinoma of
 esophagus 588

OBSTRUCTION of esophagus
 carcinoma and 526
 chronic carcinoma and 503
Oxygen after esophagectomy with esophago-
 gastric anastomosis 553

PAIN
 carcinoma of esophagus and 516 530
 foreign bodies and 459
 in peptic ulcer of esophagus 324 328
Papilloma(ta) of esophagus 491
Paralysis of esophagus 236-246
 botulism 246
 characteristics of
 clinical 237
 endoscopic 243
 roentgen 238
 diphtheritic 244
 characteristics of
 clinical 244
 roentgen 245
 prognosis in 245
 exploration in instrumental 243
 of central origin 236
 of peripheral origin 236
 prognosis in 243
 special considerations in 244
 types of 236
Parasites in esophagus 351
Paterson's method of roentgen therapy of
 carcinoma of esophagus 584
Pemphigus of esophagus 353
Perforations of esophagus *See under* Esopha-
 gus
Peristalsis esophageal 44
 waves in 72
Pentostin carcinoma of esophagus and 527
Pharyngo-esophageal junction 5
Pharynx injury to in esophagoscopy 110
Phlebotrombosis after thoracic esophagec-
 tomy 567
Phlegmon(s) of esophagus 310 *See also*
 Esophagitis phlegmonous: diffuse
 characteristics of
 clinical 312
 endoscopic, 312
 pathology of 310
 postoperative care in 314
 prognosis in 314
 treatment in 313
Phrenocardiospasm *See* Mega-esophagus
Phreno-esophageal ligament 19
Physiology of esophagus 43-51
Pilocarpine deglutition and 47
Pin(s)
 safety in esophagus extraction of 479
 straight in esophagus extraction of 476
Pinchcock
 cricopharyngeal *See* Esophagus: mouth of
 diaphragmatic. *See* Esophagus: constrict-
 tion of diaphragmatic

- Plexus
 Auerbach's 37
 Meissner's, 37
 Plummer Vinson syndrome *See* Esophagus
 Pnemonia, after thoracic esophagectomy 566
 Polyp(s) of esophagus, adenomatous 492
 Portacaval anastomosis for esophageal varices 287
 technique of, 289
 Position of esophagus
 deviation of 154
 characteristics of
 clinical 157
 roentgen 158
 treatment in 160
 Prosthesis tube for esophageal substitution 417
 Pseudodiverticula nonsphincteric 247 272
 characteristics of
 clinical 274
 endoscopic, 275
 roentgen, 274
 complications of 275
 treatment of, 275
 Pyrosis 50
- QUINIDINE after esophagectomy with esophago-gastric anastomosis 554 567
- RADON seeds in carcinoma of esophagus 579
 Reduplication of esophagus 152
 Regurgitation
 carcinoma of esophagus and 516
 foreign bodies in esophagus and 459
 persistent 460
 in mega esophagus type 1 211
 paradoxical in mega esophagus type 1 211
 Rendu Osler disease 291
 Rhabdomyoma(ta) of esophagus 496
 Rhabdomyosarcoma(ta) of esophagus 513
 Roentgen examination
 in acquired esophagorespiratory communications 429
 in acute corrosive esophagitis 365
 in Barsony-Teschendorff syndrome 185
 in benign tumors of esophagus 499
 in brachy-esophagus
 in adults 149
 in children 148
 in carcinoma of esophagus 531
 developing 517
 early 515
 terminal 521
 in chronic nonspecific esophagitis 299
 of mouth of esophagus 304
 of stenosis 382
- Roentgen examination in compression of esophagus
 by acquired abnormalities of the aorta, 172
 by congenital vascular malformations 168
 in congenital malformations of esophagus and trachea 133
 in curling of esophagus 181
 in deviation of position of esophagus 158
 in diverticulum
 of lower esophageal segment 266
 of pharyngo-esophageal segment 254
 in esophageal varices 280
 in esophagospasm in children 198
 in foreign bodies in esophagus 461
 in injuries to cervical esophagus 440
 in instrumental perforation of the esophagus 449
 in intermittent dysphagia in infants 196
 in localized parietal abscesses 309
 in mediastinitis 316
 in mega esophagus
 type 1, 213
 type 2 216
 in nonsphincteric pseudodiverticula 274
 in paralysis of esophagus 238
 botulism 246
 in peptic ulcer of esophagus 325
 in scleroderma 356
 in spasm of cricopharyngeus muscle
 chronic 193
 in spasm of epicardial segment 177 179
 chronic 194
 in spasm of pharyngo-esophageal segment 175
 in syphilis of esophagus 345
 in tuberculosis of esophagus 339
 of normal esophagus 52-79
 contrast media for 52
 positions for 53
 shadows in 53
 technical considerations in 52
 Rouget's fibers 17, 18 19
 Rupture of esophagus *See under* Esophagus
- SARCOMA(TA) of esophagus
 fibroblastic 512
 lymphoblastic 514
 osteogenic 513
 Sargnon's method of endocuretherapy 579
 Scleroderma
 esophageal changes in 353
 characteristics of
 clinical 353
 endoscopic 356
 roentgen 356
 diagnosis in differential 357
 pathology of 354
 treatment of 357
 Sengstaken-Blakemore tube 283

- Sialorrhea carcinoma of esophagus and 516
530
- Skin tube for esophageal substitution 397
- Spasm of esophagus *See* Esophagospasm
segmental *See* Barsony Teschendorff syn-
drome
- Speculum Chevalier Jackson 102
- Splenorenal anastomosis for esophageal
varices 287
technique of 288
- Sporotrichosis of esophagus 351
- Staples in esophagus extraction of 479
- Starck dilator 228
- Stenosis
cicatricial 376-396
characteristics of
clinical 381
endoscopic 382
general 379
roentgen 382
complications in 385
diagnosis in differential 384
esophageal substitution in 404
esophagoscopy in retrograde 389
etiology of 376
form of 380
lesions adjoining 380
location of 379
malignant tumors of epithelial origin
and 503
number of 380
pathology of 378
problems in special 395
prognosis in 385
treatment in 387
palliative 388
prophylactic 387
with gastrostomy 389
without gastrostomy 392
complete 127
congenital carcinoma of esophagus vs 533
partial 127
characteristics of
clinical 127
endoscopic 128
roentgen 128
diagnosis in differential 128
treatment of 128
postdiphtheritic 348
- Stomach
junction of with esophagus 20
use of for esophageal substitution 401
- Stricture of esophagus definition of 376
- Syphilis of esophagus 342
carcinoma of esophagus vs 533
characteristics of
clinical 345
endoscopic 345
roentgen 345
complications of 346
diagnosis in differential 346
pathology of 343
treatment of 346
- TAMPONADE esophagogastric 283 286
Telecurietherapy 582
Theratron external irradiation by 590
Thrush of esophagus 351
Thyroid artery inferior sympathetic plexus
of 32
- Tobacco malignant tumors of epithelial
origin and 503
- Trachea
complications involving carcinoma of
esophagus and 527
lesions of acquired esophagorespiratory
communications and 423
- Tracheostomy after thoracic esophagectomy
566
- Trauma malignant tumors of epithelial
origin and 503
- Tuberculosis
of esophagus 333
carcinoma of esophagus vs 533
characteristics of
clinical 338
endoscopic 340
roentgen 339
diagnosis in differential 340
granular 338
hypertrophic 335
pathogenesis of 333
pathology of 334
prognosis in 341
treatment in 341
types of 334
ulcerating 334
para esophageal 341
- Tumors of esophagus
benign 490-501
anatomy of pathological 490
carcinoma of esophagus vs 534
characteristics of
clinical 498
endoscopic 499
roentgen 499
diagnosis in differential 500
malignant tumors of epithelial origin
and 503
of epithelial origin 491
pedunculated 490
treatment of 501
prognosis of 500
sessile 490
treatment of 500
treatment of 500
types of 490
malignant 502-534 *See also names of
specific neoplasms*
anatomy of pathologic 504
complications of 506
etiology of 502
histopathology of 508
multiplicity of 505
of epithelial origin 502 *See also* Car-
cinoma of esophagus
age and 502

- Tumors** of esophagus, malignant of epithelial origin alcoholism and 503
 antecedent diseases and 503
 benign tumor and 503
 characteristics of, histological 508
 cicatricial stenosis and 503
 complications and 506
 esophagus and 503
 factors predisposing to 502
 heredity and, 503
 histopathology of 508
 metastatic from other organs 503
 obstruction and, chronic 503
 race and 502
 sex and, 502
 spread of, 507
 tobacco and 503
 trauma and, 503
 types of, histological 508
 of mesenchymal origin, 497, 512 *See also* Sarcoma(ta) of esophagus
 of mixed histological characteristics 514
 of nonepithelial origin 493, 504
 anatomy of, pathological 508

ULCER(s)

- contact, of mouth of esophagus 320
 characteristics of clinical 321
 treatment of 321
 peptic of esophagus 321
 characteristics of
 clinical 324
 endoscopic, 326
 roentgen, 325
 complications of 327
 diagnosis in differential 327
 esophageal substitution in 403
 pathogenesis of 322
 pathology of 323
 prognosis in 327
 treatment in 328
 surgical
 indications for, 328

- Ulcer(s) peptic of esophagus treatment in surgical results of, 332
 technique of 329

- Ulceration(s) of esophagus nonspecific 320-332

- Urticaria giant, of esophagus 352

VAGUS nerve

- components of esophagus and, 48
 course of, 36
 esophagus and 33
 interruption of esophagus and 48
 new concept of 35
 stimulation of deglutition and 47

Varices esophageal 276

- characteristics of
 clinical 280
 endoscopic 283
 roentgen 280
 diagnosis in differential 283
 etiology of 277
 pathogenesis of, 277
 pathology of 278
 prognosis in 285
 treatment in 285
 direct 285
 indirect 287
 results of 290

- Vascular ring *See* Compression of esophagus by congenital vascular malformations

Vein(s)

- abnormalities of 276-291 *See also* Varices
 portal abnormalities of 277
 splenic abnormalities of 278

- Vena cava superior abnormalities of 278

- Venous drainage of esophagus 25-27

WOOKEY esophagectomy for carcinoma of

- cervical segment 556
 complications after 563
 first stage 556
 aftercare following 560
 mortality after 564
 second stage 560

